ARCHIVES OF OTOLARYNGOLOGY

VOLUME 37 MARCH 1943 NUMBER 3

COPYRIGHT, 1943, BY THE AMERICAN MEDICAL ASSOCIATION

FACIAL PALSY OF OTITIC ORIGIN

WITH SPECIAL REGARD TO ITS PROGNOSIS UNDER CONSERVATIVE TREATMENT AND THE POSSIBILITIES OF IMPROVING RESULTS BY ACTIVE SURGICAL INTERVENTION

AN ACCOUNT OF 264 CASES SUBJECTED TO REEXAMINATION

KARSTEN KETTEL, M.D.

Chief Surgeon, Ear, Nose and Throat Department, Fredericksborg County Hospital

COPENHAGEN, DENMARK

Facial palsy is serious not because it endangers the patient directly but because, the face being "the mirror of the soul," the afflicted creature loses what Duel [1] so aptly named "the language of facial expression." That this may have far reaching psychic, social and economic consequences needs no explanation.

It is therefore understandable that in the past many attempts have been made by many different routes to come to the aid of patients with facial paralysis, but only the intratemporal surgical approach to the facial nerve seems to have brought the surgeon near the goal. Chief credit for this treatment must be given to the Englishman Sir Charles Ballance and the American Dr. Arthur B. Duel, who by their comprehensive animal experiments and subsequent operations on human beings have created the foundation on which present day surgeons build.

The results achieved by Ballance and Duel [2] and later by a whole series of others have been so brilliant that any doubt as to the value of the intratemporal operations has long since been put aside. The fact, however, remains that in a great number of cases facial paralysis due to involvement of the facial nerve in disease of the middle ear disappears after adequate treatment of the responsible aural disease, and the chief task therefore is to find means to decide which patients are the ones whose paralysis will subside spontaneously and which the ones who ought to be operated on.

Ballance and Duel [2] have been rather categoric in their demand: Every patient with facial palsy should be operated on. They expressed

1. Duel, A. B.: History and Development of the Surgical Treatment of Facial Palsy, Surg., Gynec. & Obst. **56**:382, 1933.

2. Ballance, C., and Duel, A. B.: The Operative Treatment of Facial Palsy, Arch. Otolaryng. **15**:1-70 (Jan.) 1932.

themselves thus: "The accepted time to operate is now. No delay is justifiable." And they continue:

> It has been said recently that the difficulty in deciding on operation is that we do not know whether the nerve is crushed, divided or merely involved in inflammation. We do not want to know! The question of operation has little to do with how the paralysis was produced. The reason for the operation is that the patient has facial palsy. In all early mastoid cases, inflammatory or caused by direct injury, an operation should be done immediately; the sooner it is done, the easier is the operation, the less the damage to the nerve and the better the condition of the muscles.

Tickle,[3] Duel's cooperator and heir, is somewhat less categoric, relying principally on the result of the faradic test. If the response becomes negative in forty-eight or seventy-two hours, it is a certain indication that the nerve is so severely damaged that complete spontaneous regeneration is not to be expected; on the other hand, even complete paralysis will subside spontaneously if only the reaction to the faradic test is preserved. "Those who postpone operation in cases of facial palsy with faradic response long lost remind one of the children of Ephraim, spoken of by the Psalmist, who 'being harnessed and carrying bows, turned themselves back in the day of battle'" (Ballance and Duel[2]).

The result of the faradic test has become to a growing extent the basis for deciding the question of the indication for operation in Anglo-Saxon countries. The answer might, however, be approached by another route, namely, by examining a great number of persons who were conservatively treated for otogenous facial paralysis, finding out what the results have been in the various groups and afterward trying to answer the question: Could the results have been better with surgical intervention in due time? Curiously enough, a report on reexamined materials of this kind is nowhere to be found in the available literature. To fill this gap in knowledge has been the main purpose of the present article.

METHODS OF SURGICAL INTERVENTION IN THE TREATMENT OF PERIPHERAL FACIAL PARALYSIS

When one asks whether the results could have been improved by active surgical intervention, it seems best to survey the methods at one's disposal, as then one will be able to point out the operations to be preferred in dealing with facial palsy in the various groups of cases.

Of decisive importance in choosing between the methods of operation at one's disposal is the condition of the facial muscles at the time of operation: Are they capable of contraction, or have they degenerated? Or, expressed in another way, is there a response to the galvanic test? On the whole, the answer to this question depends on the duration of the paralysis, though not entirely; for while there will

3. Tickle, T. G.: The Surgical Treatment of Facial Paralyses, in Kopetzky, S.: Surgery of the Ear, New York, Thomas Nelson & Sons, 1939.

always be response to the galvanic test at the onset of palsy, the muscles need not have degenerated and the response consequently may not be absent even if years have passed since the development of paralysis. Sullivan [4] reported paralysis of fourteen years' standing cured by nerve transplantation.

CONTRACTILE FACIAL MUSCLES

If it is possible to demonstrate by the galvanic response that the facial muscles are still contractile, all one's efforts must be aimed at supplying the paralyzed but still contractile muscles with renewed nervous impulses. The goal is (1) to restore the patient's normal appearance with the face at rest and (2) to give back to him "the language of facial expression" (Duel [1]), i. e., the emotional movements.

While the first task can be carried out in many different ways, it is possible to restore the patient's emotional facial movements, the ultimate goal of one's therapy, only if the facial nerve can be made to function again. The symmetry and the synchronism of the emotional movements of the face are dependent on close cooperation of the cortical facial centers of both hemispheres; cooperation of this kind can never be established between the facial center of one cerebral hemisphere and some different center of the other (as in anastomosis operations). Consequently the patient can regain emotional movements only if the facial nerve is repaired at the site of the lesion by an intratemporal operation, and it is this, the restoration of the symmetric emotional movements, that gives the intratemporal facial operation its advantage over all others. The latter may make the patient's face look normal at rest, and a few of them may also make it possible for the patient to perform certain voluntary movements, but they will never restore the emotional movements.

Intratemporal Facial Operations.—Under this heading are included decompression of the nerve, suture of the nerve ends and free nerve transplantation. Ney,[5] Martin [6] and Bunnell [7] devised and carried out intratemporal operations for the repair of the facial nerve, but chief credit for the enormous advance which these operations have meant to the therapy of facial paralysis is due to Sir Charles Ballance and Dr. Arthur B. Duel. The historic development has been dealt with by Duel [1] and by Tickle [8]; the technic has been described by Ballance

4. Sullivan, J. A.: A Modification of Ballance-Duel Technique in the Treatment of Facial Paralysis, Tr. Am. Acad. Ophth. **41**:282, 1936.

5. Ney, K. W.: Facial Paralysis and the Surgical Repair of the Facial Nerve, Laryngoscope **32**:327, 1922.

6. Martin, R. C.: (*a*) Intratemporal Suture of the Facial Nerve, Arch. Otolaryng. **13**:259 (Feb.) 1931; (*b*) Surgical Repair of the Facial Nerve, ibid. **23**:458 (April) 1936; (*c*) Recent Experiences with Operation on Facial Nerve, ibid. **32**:1071 (Dec.) 1940.

7. Bunnell, S.: Surgical Repair of the Facial Nerve, Arch. Otolaryng. **25**: 235 (March) 1937.

and Duel [2] and by Tickle [3]; these aspects will therefore be only touched on in the following pages.

(*a*) Decompression: In this operation the canal for the facial nerve is opened from the stylomastoid foramen to the level of the horizontal semicircular canal, and the nerve sheath is slit open with the object of relieving pressure on the nerve (from edema, hemorrhage or bone spicule), which lies encased in a firm tube with unelastic walls; this pressure, if of sufficient intensity and duration, may cause the nerve to degenerate. Decompression is also preliminary to suture of nerve ends and to free nerve transplantation.

A number of writers have successfully employed decompression for otogenous (Ballance and Duel [2]; Cawthorne; Kettel [8]; Martin [6]; Morris [9]; Sewall [10]; Tickle [3]; Watson-Williams [11]) and for rheumatic (Ballance and Duel [2]; Cawthorne; Kettel [8]; Morris [9]; Sandor [12]; Tickle [3]) facial palsy.

(*b*) Direct Suture of the Nerve Ends: If the continuity of the nerve has been interrupted and the gap is not more than 1 or 2 mm., direct suture of the nerve ends can be done, the nerve remaining in its canal. If, however, the gap is wider, it is possible, as described by Bunnell,[7] to shorten the course of the nerve as much as 23 mm. by rerouting, i. e., lifting the nerve out of its canal and altering its course (see *A* in figure). In this way a number of writers (Bauer [13]; Bergström [14]; Kettel [8]; Martin [6]; Ney [5]) have achieved excellent results. Direct suture in Bunnell's [7] opinion is superior to transplantation, while Ballance and Duel [2] prefer free nerve transplantation to suture by rerouting.

(*c*) Free Nerve Transplantation: If the diastasis is too wide to be overcome by suture, the proximal and distal segments of the divided facial nerve can be united by free nerve transplantation (*B* in figure). For this purpose the nervus cutaneus femoris anterior is used as being of a fitting caliber and easily accessible. The length of the graft

8. Kettel, K.: Surgical Treatment of Peripheral Facial Paralysis, Ugesk. f. læger, to be published.

9. Morris, W. M.: Surgical Treatment of Facial Paralysis: A Review of Forty-Six Cases, Lancet **2**:558, 1939.

10. Sewall, E. C.: Decompression of the Facial Nerve, Arch. Otolaryng. **18**: 746 (Dec.) 1933.

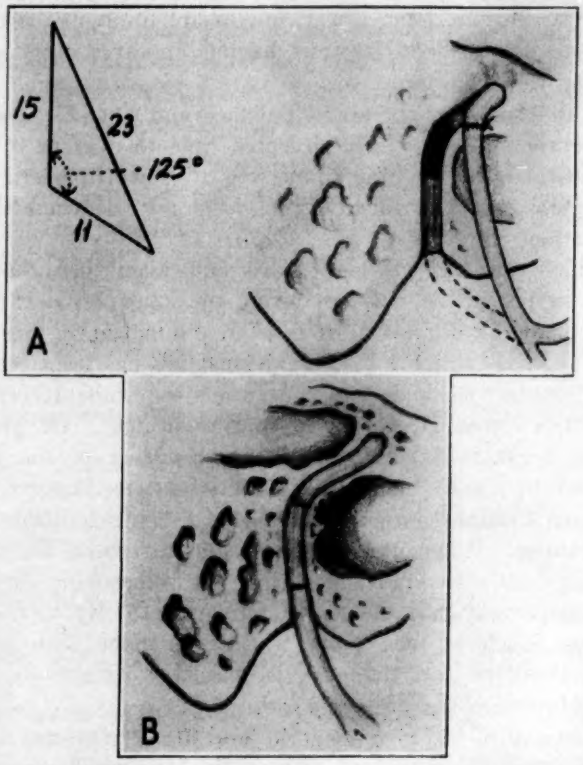
11. Watson-Williams, E.: Facial Paralysis Occurring During Mastoid Operation; Decompression of Facial Nerve; Complete Recovery, Proc. Roy. Soc. Med. (Sect. Otol.) **22**:98, 1929; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **14**: 856, 1930.

12. Sandor: Two Cases of Facial Palsy "e Frigore" Cured by Decompression, J. Laryng. & Otol. **49**:503, 1934.

13. Bauer, G.: Nerve Graft in Facial Palsy, Acta chir. Scandinav. **81**:130, 1938.

14. Bergström, E.: Good Result After Operation for Traumatic Defect of the Facial Nerve, Nord. med. (Hygiea) **8**:2102, 1940.

has varied from a few to more than a hundred millimeters (Hall [15]). Cardwell [16] has even successfully used a graft of such length that the distal end was implanted directly into the paralyzed muscles. Tickle [3] in 1 case in which the geniculate ganglion was involved in the destructive process succeeded in uniting the proximal end of the facial nerve just below where it pierces the dura with the distal end by means of a 16 mm. graft. If this procedure is impossible, it is necessary, when the nerve has been injured so near the brain, to make an anastomosis with the accessory or hypoglossal nerve (Poppen [17]; Eagleton [18]).



A, suture of the facial nerve after the method described by Bunnell [7]; *B*, nerve graft after the method of Ballance and Duel.[2]

15. Hall, I. S.: Two Cases Illustrating Ballance-Duel Technique, J. Laryng. & Otol. **52**:648, 1937; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**: 672, 1938.

16. Cardwell, E. P.: Direct Implantation of Free Nerve Grafts Between Facial Musculature and Facial Trunk, Arch. Otolaryng. **27**:469 (April) 1938.

17. Poppen, cited by Eagleton.[18]

18. Eagleton, W. P.: Intradural Conditions in Relation to Rhinology and Otology: Critical Survey of Literature, Arch. Otolaryng. **32**:109 (July) 1940.

The graft serves as a guide for the axons of the proximal nerve end, which grow along it into the distal part of the nerve and from there onward along the peripheral ramifications into the muscles. Instead of one single graft Bunnell and Boyes [19] recommended "cable grafts." These writers showed on the strength of animal experiments that a single thick graft is more inclined to necrotize as compared with several grafts of less caliber, in which more tissue is kept alive. A long time will now have to pass before the muscles begin to function again; Tickle [8] estimated this period as at least eight months; Morris,[9] six to twelve months; I had to wait fourteen months in a case in which the graft was 35 mm. long, a wait unpleasant for both the patient and the surgeon. But, the first signs having appeared, further mobility will develop rapidly.

When is it time to operate? Ballance and Duel [2] expressed their opinion clearly enough: "The accepted time to operate is now. No delay is justifiable." In this connection it is worth bearing in mind that in their opinion suppuration does not contraindicate nerve transplantation.

As after-treatment, massage is recommended, because furthering the blood supply of the muscles during the long period of waiting, in order to counteract degeneration, is of first importance. As to electric treatment opinions differ. While galvanization has been recommended by many writers, Ballance and Duel [2] advised some reservation, and Morris [9] stated that electricity is contraindicated. Of great benefit during the night is a hook placed in the corner of the mouth and held in place by a piece of string or adhesive plaster fastened in front of the ear; this is claimed to prevent strain of the muscle fibrils during the time of waiting. When mobility has begun to return, the patient will have to carry out active movements in front of a looking-glass. This is the most important part of the after-treatment. By this exercise he learns how much he can move at any moment with preservation of facial symmetry and thus avoids the large excursions, which the affected side cannot completely equal.

It is seen that the intratemporal operations are ideal in principle because, as already emphasized, only in this way can the patient's emotional movements be restored; but how great an effect will intratemporal operation have on the facial nerve?

By decompression it is possible to obtain full function of the nerve, a result not reached by even the most successful suture or nerve transplantation. Tickle [3] expressed the belief that even the best result of either of the two last-mentioned operations can at most be estimated at 85 per cent of normal function. If, however, one keeps in mind that these operations are carried out on patients who without operation

19. Bunnell, S., and Boyes, J. H.: Nerve Grafts, Am. J. Surg. **44**:64, 1939.

are condemned to lead a life under the curse of facial palsy, and if one looks at the whole series of brilliant results reported (Bauer [13]; Bergström [14]; Brooke [20]; Bunnell [7]; Cawthorne; Collier [21]; Kisch [22]; Kettel [8]; Lindsay [23]; Martin [6]; McArthur [24]; Scott [25]; Sullivan [4]), one easily realizes what enormous progress has been made through the fundamental work of Ballance and Duel.[2] The value of the intratemporal operations should not be measured by how much the final result lacks of being 100 per cent function but by how much has been attained compared with the condition that would have been present without surgical intervention.

If the muscles are contractile but not one of the intratemporal operations can be used (a rare occurrence), attempts must be made to supply the muscles with renewed impulses by other methods. This end is served by neurotization and anastomoses.

Neurotization.—By this is understood attempts to make the neurofibrils from pedicle muscle flaps (masseter and temporal muscles) grow into the paralyzed muscles. The method has been worked out especially by Rosenthal [26] and Doroschenko [27] and has also been used by Cistjakov [28] and Jaritzin,[29] and these writers have achieved a series of acceptable results. All told, however, the method has proved a disappointment: The face can be made symmetric in repose, the patient can be enabled to carry out certain voluntary movements, but the emotional movements—the most important thing—remain lost.

---

20. Brooke: A Case of Facial Paralysis Treated by Facial Grafts, Brit. J. Surg. **20**:523, 1933.

21. Collier, D. J.: Facial Paralysis: Ballance-Duel Nerve Graft, Proc. Roy. Soc. Med. **29**:1679, 1936.

22. Kisch, H.: Facial Paralysis: Ballance-Duel Nerve Graft; Recovery, Proc. Roy. Soc. Med. **29**:1679, 1936; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**:54, 1938.

23. Lindsay, J. R.: Transplantation of Facial Nerve, Arch. Otolaryng. **27**: 131 (Jan.) 1938.

24. McArthur, G. A. D.: Repair of Facial Nerve Lesion by Nerve Graft, M. J. Australia **2**:1123, 1939.

25. Scott, S.: Nerve-Graft in the Treatment of Facial Paralysis: Ballance and Duel Method, Proc. Roy. Soc. Med. **28**:236, 1935; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **25**:61, 1936.

26. Rosenthal, W.: Ueber muskuläre Neurotisation bei Facialislähmungen, Zentralbl. f. Chir. **43**:489, 1916.

27. Doroschenko, S. T.: Verbesserungen bei der Paralyse des Gesichtes, Monatschr. f. Ohrenh. **70**:1303, 1936.

28. Cistjakov: Ueber operative Behandlung der Fazialisparalyse mittels Muskelplastik nach Rosenthal, Nov. khir. **12**:222, 1931; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **18**:110, 1932.

29. Jaritzin, A. A.: Muscle Neurotisation in the Case of Facial Palsy, Folia neuropath. eston. **15-16**:131, 1936; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **27**:226, 1937.

Anastomoses.—From the close of the previous century operations of this type were in rather extensive use until the arrival of the intratemporal operation on the facial nerve (a detailed bibliography is to be found in the articles of Eden [30] and Nühsmann [31]).

The facial nerve has been anastomosed with a number of cranial nerves: nervus accessorius (most often), nervus glossopharyngeus, nervus hypoglossus and nervus descendens noni. In this way, symmetry can be procured for as long as the face remains at rest, and the patients may be enabled to perform some voluntary movements; the emotional ones, however, will still be lacking. Add to this that the results are marred by associated movements: In the case of anastomosis with the accessory nerve violent twitchings are no rare occurrence when the shoulder is being raised; in the case of anastomosis with the hypoglossal or the glossopharyngeal nerve the same thing is in evidence during eating and drinking. A further drawback is the appearance of deficiency phenomena; thus in the case of an anastomosis with the accessory nerve paresis and atrophy of the shoulder muscles are observed. On the whole the anastomosis must be looked on as a past stage in the development of therapeutics; cure should not be sought by "the run-about-way of anastomosis" (Ballance and Duel [2]).

DEGENERATED FACIAL MUSCLES

If the facial muscles have degenerated and the galvanic response is consequently missing, the intratemporal operations are contraindicated. In this case efforts must be aimed at lifting the paralyzed corner of the mouth and diminishing the palpebral fissure. Gersuny injected iron chloride in order to produce an aseptic inflammation with cicatricial contracture following. Momburg [32] lifted the corner of the mouth by means of a subcutaneously placed thread of aluminum-bronze, while Moskowicz,[33] Kirschner [34] and Demel [35] employed a strip of fascia lata for the same purpose. By the methods mentioned here the corner of the mouth is fixed in a fitting position; to obtain some vol-

30. Eden, R.: Ueber die chirurgische Behandlung der peripheren Fazialislähmung, Beitr. z. klin. Chir. **73**:116, 1911.

31. Nühsmann, T.: Diagnose, Prognose und Therapie der otogenen Fazialislähmung, in Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 8, pt. 3.

32. Momburg: Die kosmetische Behandlung der Fazialislähmung nach Busch, Berl. klin. Wchnschr. **47**:1115, 1910.

33. Moszkowicz: Faszienplastik bei Fazialislähmung, Wien. klin. Wchnschr. **41**:1151, 1928.

34. Kirschner, M.: Die gegenwärtige Stand und die nächsten Aussichten der autoplastischen freien Fascienübertragung, Beitr. z. klin. Chir. **86**:45, 1913.

35. Demel, R.: Ein Beitrag zur Fascienplastik bei Facialislähmung, Zentralbl. f. Chir. **61**:1445, 1934.

untary mobility, however, some writers (Brunner; Eden [30]; Gillies [36]; Gomoin [37]; Halle [38]; Lexer [39]; Perthes [40]; Sanvenero-Roselli [41]) have carried out myolastic operations, fixing pedicle strips of the masseter and temporal muscles in the nasolabial groove and in the palpebrae (as in neurotization, even if the operation was intended only to provide support for the angle of the mouth, not to procure reinnervation). Gillies [36] and Halle [38] have gone a step further, isolating some bundles of the temporal muscle, extending these by fascial strips and fixing them by suture in the nasolabial groove. All these procedures are, however, as Halle [38] also emphasized, only makeshifts in cases in which it is impossible to supply the facial muscles with fresh nerve impulses.

In order to diminish the palpebral fissure partial tarsorrhaphy has been performed, and operations have been done on the superior cervical ganglion and the corresponding rami communicantes (Leriche and Hesse [42]) with the object of producing some degree of enophthalmos.

DESCRIPTION OF MATERIAL AND ANALYSIS OF OBSERVATIONS ON REEXAMINATION

The material was compiled in the department of otolaryngology of the Municipal Hospital (chief surgeon S. H. Mygind, M.D.), in Copenhagen. The patients of this hospital are recruited in the main from the working classes of the capital. During my time as assistant surgeon of the department I collected and examined the material, and I have permission from my former chief to use it. The case records of all patients with peripheral facial palsy caused by acute suppurative otitis media admitted between the time of the inauguration of the department, in 1906, and Jan. 1, 1938 have been included. During these thirty-two years there were treated in the indoor department 13,135 patients with suppurative otitis media, acute or chronic, 264 of whom had peripheral facial palsy. Excluded are 2 patients whose palsy was supposed to be of central origin and 2 in whom a tumor was the actual cause.

36. Gillies, H.: Experiencs with Fascia Lata Grafts in the Operative Treatment of Facial Paralysis, J. Laryng. & Otol. **49**:743, 1934.

37. Gomoin, cited by Eden.[30]

38. Halle, M.: The Occurrence of Facial Paralysis and Methods for Its Correction, Laryngoscope **48**:225, 1938.

39. Lexer, E.: Zur Gesichtsplastik, Arch. f. klin. Chir. **92**:786, 1910.

40. Perthes: Ist die Nervenpropfung oder die Muskelplastik für die Behandlung irreparabler Facialislähmungen vorzuziehen? Zentralbl. f. Chir. **51**:2073, 1924.

41. Sanvenero-Roselli, G.: Paralisi del facciale e sua cura chirurgica, Atti clin. otol., Univ. Torino **2**:299, 1935.

42. Leriche and Hesse, cited by Doroschenko.[27]

The periods during which the patients were observed may be seen in table 1. No patient was observed for less than one year, and 153 of 169 patients were under observation for more than three years, i. e., for a period long enough to preclude with certainty any possibility of later spontaneous recovery.

To make reexaminations possible, all patients were invited by letter as many as three times to appear at the hospital for examination. Fifty-four patients who did not appear I sought out and examined at their homes. In spite of tenacious utilization of all available means, including the Salvation Army, I succeeded in finding only 244 of the 264 patients with facial palsy (92.4 per cent), partly because the Population Register was not set up until 1918, whereas the material dates back as far as 1906, and partly because the dating of the case records was often insufficient.

All patients found still alive in this country have been reexamined. Of the 264 patients, 43 had died in the hospital (30 of labyrinthogenous meningitis, 6 of extralabyrinthine meningitis, 3 of pulmonary complications, 2 of septicemia and 2 of deep osteitis), 29 had died later from

TABLE 1.—*Grouping of Cases According to Observation Periods*

Observation time, yr.	1	2	3	4	5	6-10	11-20	21-30	Over 31
Cases	4	12	10	14	8	28	58	23	12

unknown causes, 3 were living abroad and 169 were living in this country. This leaves 20 patients whose fate is unknown.

A detailed form was filled in, to secure thoroughness and uniformity, and the reexaminations with few exceptions were carried out by me, a uniform evaluation thus being insured. In the records the word "paresis" had been used as synonymous with "paralysis"; the additional "partial" could not always be defined clearly, as in many cases the notations did not show exactly what was meant: a partial paresis of all branches or a complete paralysis of one branch only (e. g., the oral branch, the function of the others being intact).

The patient's face was examined at rest and during facial movements. When the face was examined in repose, immediately it struck the eyes that by far the greatest part of the palsy had, after some years' duration, apparently disappeared spontaneously. By functional investigation, however, a more or less pronounced remainder was demonstrated.

The reexamined patients can be divided into three main groups: (1) those whose paralysis had completely disappeared, (2) those whose paralysis had apparently disappeared, being in evidence only during movements of the facial muscles, not in repose, and (3) those whose paralysis had left its distinct marks on the face even at rest. The majority of the conservatively treated patients fell into groups 1 and 2.

OBSERVATIONS ON REEXAMINATION

The functions of the three branches of the nerve were examined separately:

The Mouth.—In most cases the angle of the mouth drooped somewhat on the paralyzed side as compared with the unaffected side but not infrequently it was on exactly the same level, even when it was completely immobile. In some cases a contracture had developed so that the affected angle was raised, but this was decidedly exceptional. That the mouth was on only a slightly lower level on the paralyzed side, if not symmetric, is undoubtedly explained by the fact that even the most slender connection between the nerve segments above and below the site of injury is sufficient for preserving the tone of the oral muscles. Of this I have recently been convinced during decompression and transplantation; in spite of the fact that the angle of the mouth was completely immobile, it was on the same level on the paralyzed side as on the unaffected side in these cases.

If patients with peripheral facial paralysis are examined some years after the appearance of the palsy, a maximal droop of the affected side of the mouth is seen only in those in whom the conduction of nerve impulses has been completely interrupted. If only the slightest connection remains between the proximal and the distal part of the nerve, this suffices to keep the oral muscles at a degree of tone that enables the angle of the mouth on the defective side to be on about the same level as the angle on the unaffected side.

If the fact that the angle of the mouth was in most cases on the same level on both sides were due alone to contracture on the paralyzed side, it seems peculiar indeed that the only patients with a real droop of the mouth were those in whom the continuity of the facial nerve had with absolute certainty been broken; here contracture, one would think, might equally well have been in evidence.

Judging by the information contained in the case records and obtained from the patients on reexamination, the branch to the muscles of the mouth seems to be the one that regenerates best and most rapidly. Morris [9] has had the same experience.

The Eye.—In cases of fresh, complete paralysis, the eye cannot be closed, and at the same time Bell's phenomenon is present; i. e., the observer is able to see that the eyeball is rolled upward and outward during the attempt at closing the eye. An apparent closing is effected by relaxation of the levator palpebrae superioris. Fowler Jr.[43a] pointed

43. Fowler, E. P., Jr.: (*a*) Abnormal Movements Following Injury to the Facial Nerve, J. A. M. A. **113**:1003 (Sept. 9) 1939; (*b*) The Management and Treatment of Afflictions of the Facial Nerve Within the Fallopian Canal, Acta oto-laryng. **27**:615, 1939.

out that at a later time it may be difficult to determine whether the closing of the eye is due to "partial repair of the branch of the VIIth nerve to the orbicularis oculi or to increased education of some other nerve, probably the IIIrd. This movement of the upper lid will sometimes permit the eye to close with complete paralysis of the facial nerve."

Van Gielse [44] pointed out that the blinking reflex is the last movement to disappear and the first to return. If one watches a patient with apparently complete facial paralysis, one sometimes sees rapid horizontal movements along the edge of the lower lid toward the nose. This is said to be a favorable prognostic sign. However, if these movements have been present but have disappeared again, the outlook is bad. As long as the blinking reflex is still present, however small the excursions of the lower lid, Bell's phenomenon will be absent. While a continuous flow of tears was a frequent occurrence, necessitating partial tarsorrhaphy in 2 patients, I did not find ulcerations on the eyeball in a single case.

The Forehead.—The muscular functions of the forehead return last if they return at all. In numerous cases the reexamination revealed that the mouth functioned beautifully and that the eye could be closed, while the forehead remained completely immobile, and exactly the same result has been experienced in nerve transplantation and in suture of the nerve. The explanation is supposed to be that the fewer contractile elements present in the frontalis muscle degenerate before innervation has been reestablished (Martin [6b]). Only in a single case has function of the frontalis muscle been seen (by Martin [6c]) to return after free nerve transplantation.

Two phenomena will show that a patient has had facial palsy, even if no remainders of it can be seen either with the face at rest or during facial movements. One is synkinesis; the other is lowered faradic irritability. Synkinesis is associated movement: If a patient tries, for example, to perform isolated movements within the facial region supplied by one of the three branches of the facial nerve, movement is automatically induced also within another region. An attempt to blink or close the eye will provoke concomitant twitching at the corner of the mouth, more or less marked, but often strongly pronounced. Fowler Jr.[43a] went further into this question of facial synkinesis and on the strength of experimental investigations by Howe, Tower and Duel [45] explained them in the following way: The three branches of the facial

44. van Gielse, P. H. G.: Le clignement dans la paralysie faciale périphérique otogène, Acta oto-laryng. **24**:162, 1936.

45. Howe, H. A.; Tower, S. S., and Duel, A. B.: Facial Tic in Relation to Injury of Facial Nerve: Experimental Study, Arch. Neurol. & Psychiat. **38**:1190 (Dec.) 1937.

nerve lie isolated within the sheath of the main trunk, separated by connective tissue (as shown by Hofmann [46]). If the nerve is severely damaged, on an attempt at regeneration every single axon in the proximal nerve segment will split into several neurofibrils. These seek to penetrate to the corresponding muscle via the distal portion of the nerve, but in this attempt not all fibrils originating from one single axon, let us say the trunk to the eye, succeed in reaching their proper destination, the distal nerve end where they "belong," some of them growing into and along the branches innervating the oral or the frontal muscle. If now impulses are conveyed through the axon, which consists of several neurofibrils, movements will result not only in the region formerly innervated by this axon but in all regions to which its ramifications have penetrated. During attempts at closing the eye movements will arise around the corner of the mouth (that there will be no movements of the frontal muscle will be explained soon). The more severe the lesion of the nerve, the stronger as a rule will be synkinesis, but not infrequently the only remainder of paralysis to be observed, if attention is paid to this phenomenon, is weak synkinesis. My investigations have shown that synkinesis is absent only when the damage to the nerve has been so slight that the axons have, supposedly, not degenerated and therefore not split, the precondition of synkinesis being thus lacking, or when the damage has been so extensive that the nerve has been totally interrupted, the neurofibrils thus being unable to ramify to the portion of the nerve peripheral to the site of injury. In a few cases tic may be observed as a consequence of paralysis. According to my investigations, however, this is rather rare. The explanation of the phenomenon given by Ballance and Duel [2] is that normally a continuous current is passing through the nerve keeping the muscles in tone. If the nerve is damaged, the site of injury provides a hindrance to this normal continuous flow of impulses. These are being held up on the proximal side of the lesion to break through the barrier suddenly when they have gained sufficient accumulated strength, the result being ticlike contractions.

Sequelae of an apparently completely vanished facial paralysis can be demonstrated through examination of the faradic irritability of the nerve. This shows invariably a reduction fairly proportional to the degree of paralysis, and, as already mentioned, it has been possible in some cases to demonstrate slight diminution of the faradic response as the only vestige of the former paralysis.

The Sense of Taste.—Finally the sense of taste was examined as to the qualities salt, sweet, sour and bitter on the anterior two thirds of the tongue in 111 patients.

---

46. Hofmann, L.: Der Faserverlauf im Nervus facialis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **10**:86, 1924.

Examinations of taste in patients with abnormal conditions of the ear have in more recent times been performed by Szende,[47] Wohlfeil[48] and Ho.[49] The last found no disturbances of taste in cases of acute otitis, whereas taste was totally lost in the anterior two thirds of the tongue innervated by the chorda tympani in 17 patients who had had a radical operation on the ear. Wohlfeil found taste lacking within the region of the tongue just defined in 25 patients who had had a radical operation, while in 2 patients it was found normal. The majority of these patients, however, were examined shortly after their operation. Special interest is attached to investigations by Szende.[47] This writer divides his patients into two groups: those examined less than three years and those examined more than three years after operation. In the first group of patients taste was completely lost, while in the second normal

TABLE 2.—*Observations on the Sense of Taste on the Anterior Two Thirds of the Tongue in Cases of Paralysis After Three or More Years*

Preoperative paralysis in connection with:	Cases	Taste on Affected Side
Acute otitis media	23	Normal
	2	Lowered
Chronic otitis media	17	Normal
	7	Lowered (in 1 patient totally lost)
Paralysis following:		
Simple mastoidectomy	12	Normal
	5	Lowered
	1	Intensified
Radical mastoidectomy and labyrinthectomy	39	Normal
	4	Lowered
	1	Intensified

taste was demonstrated. To explain this phenomenon Szende presumed that taste was taken over partly by the chorda of the opposite side and partly by the glossopharyngeal nerve of the same side, which ramified directly toward the tip of the tongue. My own investigations confirm Szende's results. They were carried out with the technic of the writers mentioned in this paragraph, taste being tested as regards salt, sour, sweet and bitter by means of solutions of sodium chloride (10 per cent), acetic acid (1 per cent), sugar (5 per cent) and quinine sulfate (1 per cent). Only patients observed for three years or more are included in the report of results given in table 2.

The tests revealed normal taste in practically all patients with preoperative facial palsy in connection with acute otitis media; in only 2

47. Szende: Untersuchungen über die Geschmacksempfindungen nach Ohroperationen, Monatschr. f. Ohrenh. **69**:737, 1935.

48. Wohlfeil, M.: Ueber Geschmacksstörungen nach Mittelohrerkrankungen und Radikaloperationen, Inaug. Dissert., Königsberg, 1936.

49. Ho, W. Y. H.: Disturbances of Taste of Otitic Origin with Special Reference to Operations on the Ear, Arch. Otolaryng. **26**:146 (Aug.) 1937.

of the 25 patients with acute otitis media was taste reduced, and these 2 patients also showed permanent paralysis of the motor branches of the facial nerve. Three fourths of the patients with preoperative facial palsy in connection with chronic otitis media showed normal taste on being tested a number of years later. Two patients in this group revealed a dissociated sense of taste; perceptibility in one patient was natural only for sweet, being lowered for the three other qualities; the other patient perceived sour with definitely increased intensity but showed no divergence from normal with respect to the remaining three qualities. As

TABLE 3.—*Total Material*

Variety of Suppurative Otitis Media	Cases	Number with Preoperative Facial Paralysis	Percentage	Number with Postoperative Facial Paralysis	Percentage	Number with Postoperative and Preoperative Facial Paralysis	Percentage
Acute	9,891	50	0.5	46	0.47	96	0.97
Chronic	3,244	55	1.7	113	3.50	168	5.20
Total	13,135	105	0.8	159	1.20	264	2.00

TABLE 4.—*Postoperative Paralysis*

Operation	Cases	Number with Paralysis	Percentage
Resection of the mastoid process	4,797	37	0.7
Resection of the mastoid process and of the tympanic cavity	1,625	70	4.3
Labyrinthectomy	218	50	23.0
Total	6,640	157	2.4
Removal of granulations	.....	1	....
Removal of polyps of the middle ear	.....	1	....

regards postoperative paralysis, my investigations confirm the observations of Szende,[47] as nearly all the patients (39 of 44) who had had a radical operation, on being tested more than three years later had a normal sense of taste, which Szende [47] ascribed, as mentioned earlier, to the fact that the chorda tympani of the opposite side or the glossopharyngeal nerve of the same side had taken over the function. Regeneration of the chorda may be looked on as impossible.

FREQUENCY OF OTOGENOUS FACIAL PARALYSIS IN CASES OF OTITIS MEDIA

The frequency of facial paralysis in connection with acute and with chronic otitis media and with operations for the treatment of these conditions is shown in tables 3 to 5. In tables 3 and 4 my own material

is shown, and in tables 5, 6 and 7 comparisons are made with the only material of some size published. A more detailed study of these tables reveals the following facts:

1. The total number of patients with facial palsy (preoperative and postoperative) is found by all three authors to be 2 per cent of the total number of patients with otitis media, acute or chronic; if one con-

TABLE 5.—*Comparison of the Material of Various Writers*

Writer	Cases of Acute and Chronic Otitis Media	Number with Preoperative Facial Paralysis	Number with Postoperative Facial Paralysis	Total Number with Facial Paralysis	Percentage
Persky [51]	1,500	20	11	31	2.0
Pöllmann [50]	2,768	41	24	65	2.3
Kettel [8]	13,135	105	159	264	2.0

TABLE 6.—*Comparison Between Pöllmann's and Kettel's Material: Preoperative Palsy*

	Acute Suppurative Otitis Media			Chronic Suppurative Otitis Media			Acute and Chronic Suppurative Otitis Media		
Writer	Cases	Number with Palsy	Percentage	Cases	Number with Palsy	Percentage	Cases	Number with Palsy	Percentage
Pöllmann [50]	1,824	14	0.7	944	27	2.9	2,768	41	1.5
Kettel [8]	9,891	40	0.5	3,244	55	1.7	13,135	105	0.8

TABLE 7.—*Comparison Between Pöllmann's and Kettel's Material: Postoperative Palsy*

	Resection of the Mastoid Process			Resection of the Mastoid Process and of the Tympanic Cavity			Labyrinthectomy		
Writer	Cases	Number with Palsy	Percentage	Cases	Number with Palsy	Percentage	Cases	Number with Palsy	Percentage
Pöllmann [50]	1,812	12	0.66	623	7	1.1	18	5	27.7
Kettel [8]	4,797	37	0.77	1,625	70	4.3	218	50	23.0

siders the widely differing sizes of the material, the accordance is almost amazing.

2. Among patients with acute suppurative otitis media those with facial paralysis amount to 1 per cent, the number with preoperative paralysis being about the same as the number with postoperative paralysis (table 3).

3. In cases of chronic suppurative otitis media facial palsy is a much more frequent complication than it is in cases of acute otitis media, being demonstrated in 5 per cent of the cases (table 3), preoperative in

one third and postoperative in the remaining two thirds. This has been stressed also by other writers (Nühsmann [31]; Pöllmann [50]).

4. Preoperative facial paralysis is three times more frequent in cases of chronic than in cases of acute suppurative otitis media (table 3). This finds its natural explanation in the pathologic changes, the paralysis occurring with acute suppurative otitis media being of toxic origin, as will be shown later, while that occurring with chronic suppurative otitis media is due as a rule to involvement of the nerve by the osteitic process. The cases of preoperative paralysis in my material amount to 0.8 per cent (105 of 13,135); those in the material of Pöllmann [50] to 1.5 per cent (41 of 2,768), and those in the material of Persky [51] to 1.3 per cent (20 of 1,500). This difference probably may be ascribed to the dissimilarity of the materials; Pöllmann expressly pointed out that the number of instances of facial palsy in his material had been dwindling rapidly in recent years, since the patients were being subjected to early treatment. Persky's material includes only cases of facial palsy in connection with osteitis of the mastoid, none in connection with simple acute otitis media; i. e., it is a sorted material, and consequently his figures are somewhat higher than mine, which include the latter group. The total figures are so small, however, that this in itself accounts for imaginary differences.

5. Postoperative facial paralysis is seven times more frequent in cases of chronic otitis media than in cases of acute otitis media (table 3). This is explained partly by the pathologic changes, partly by the fact that in cases of chronic otitis media one has to perform operations which experientially involve greater risks of injury to the nerve (radical mastoid operation, labyrinthectomy). Table 7 shows a comparison between Pöllmann's and my data on the occurrence of postoperative paralysis, and while there is no great difference as regards simple mastoidectomy and labyrinthectomy, I have no less than four times as many cases in which postoperative paralysis followed a radical mastoid operation. This is probably explained by the difference in material. If, for example, the proportion of radical mastoid operations to labyrinthectomies is regarded, it will be seen to be as 35 to 1 (623 to 18) in Pöllmann's material and as 7 to 1 (1,625 to 218) in mine, i. e., apparently his cases were far more serious. To some extent the explanation of this may possibly be that the patients have come under treatment at varying times; if the cases with preoperative and those with postoperative paralysis are taken together, Pöllmann has among 944 cases of chronic otitis media $27 + 12 = 39$ with paralysis, or 4.1 per cent; the corresponding figures

50. Pöllmann: Fazialisparesen, Monatschr. f. Ohrenh. **71**:1068, 1937.

51. Persky, A. H.: Facial Palsy of Otitic Origin, Arch. Otolaryng. **27**:395 (April) 1938.

in my material are 55 + 113 = 168 among 3,244 cases of chronic otitis media, or 5.2 per cent. Thus, we have fairly similar total figures in regard to the occurrence of paralysis with chronic suppurative otitis media, including both preoperative and postoperative paralysis. Among 339 of Körner's [52] patients who had a radical operation there were 17 with preoperative and 15 with postoperative facial palsy, or 4.4 per cent.

6. The enormous significance of cholesteatoma in the genesis of facial palsy appears from table 8. The total number of cases in which facial palsy occurred in the material was 264, of which 123 were cases of cholesteatomatous otitis media, i. e., 47 per cent. In no less than about half the total number of cases in which facial paralysis was seen in the department it was due to cholesteatomatous suppuration, and particularly clear was the enormous significance of the acute exacerba-

TABLE 8.—*Frequency of Facial Palsy Associated with Cholesteatomatous Otitis Media*

Chronic Otitis Media	Number in Which There Was Acute Exacerbation of Cholesteatomatous Otitis Media	Number in Which There Was Chronic Cholesteatomatous Otitis Media	Total	Percentage
55 cases in which preoperative palsy occurred	40	4	44	80
113 cases in which postoperative palsy occurred	65	14	79	70
168 cases in which postoperative and preoperative palsy occurred	105	18	123	73

tion of chronic cholesteatomatous suppuration for the appearance of facial palsy.

PROGNOSIS AND TREATMENT IN THE VARIOUS GROUPS OF CASES INVESTIGATED

PREOPERATIVE PARALYSIS

Acute Suppurative Otitis Media.

Pathogenesis.—Björk [53] described how the nerve can be injured anywhere in its course from the porus acusticus internus to the foramen stylomastoideum.

At the porus acusticus internus the nerve may be affected by an extradural abscess. During its course through the petrous bone it may become involved in

52. Körner, O.: Ueber Fazialislähmung infolge von Operationen im Mittelohr und am Schläfenbein, Ztschr. f. Hals-, Nasen- u. Ohrenh. **72**:186, 1915.

53. Björk, H.: Peripheral Facial Paralysis Following Acute Otitis, Finska läk.-sällsk. handl. **79**:622, 1936.

an inflammatory process present in the perilabyrinthine cells or in the interior of the labyrinth. Particularly exposed is the nerve in its close relation to the middle ear where it bends around the vestibular fenestra. In this place the wall of the canal for the facial nerve is thinnest, and very often physiologic dehiscences are present here. This is best understood when it is remembered that in the embryo the channel for the nerve develops as a groove which only later is transformed into a canal. To these dehiscences some writers attach great importance as regards the genesis of facial paralysis, while others consider them of no significance. In its descending course the facial nerve may become involved in an inflammation of the retrofacial cells; or the inflammation may reach the nerve by penetrating the posterior wall of the external auditory meatus. It is also worth keeping in mind that sometimes, as a consequence of developmental disturbances, there persists a canal which issues from the hypotympanum and crosses the canal of the facial nerve on its way toward the apex of the mastoid process. In that canal the nerve may lie exposed. Finally, at its exit from the foramen stylomastoideum, it is exposed to both osseous and nonosseous processes.

By a thorough neurologic examination one is able to determine exactly where in its course the nerve has been damaged (Bunnell [7]; Nühsmann [31]; McCaskey [54]; Morwitz [55]; Lillie [56]).

Preoperative facial paralysis is partly primary (most often)—in that case it is due either to toxic neuritis or to edema in the loose connective tissue surrounding the nerve, which is compressed on account of the inelasticity of the bony canal—and partly secondary, osteitis extending to the nerve, which is consequently exposed to granulating inflammation.

The pathogenesis was dealt with in detail by Pollak,[57] who pointed out the significance of the extremely thin osseous wall which separates the nerve from the middle ear and the numerous vascular anastomoses between the latter and the canal. In his opinion the paralysis is not due to direct contagion but to irritative processes in the neurovascular apparatus that are dependent on the middle ear suppuration. The vasomotor nerves to the vessels connecting the middle ear with the canal become the victims of toxic paralysis, the consequence being edema, possibly followed by degenerative changes in the nerve.

The treatment of the preoperative facial palsy complicating acute otitis media in my material has been myringotomy in 9 cases, mastoidec-

54. McCaskey, C. H.: The Etiology and Treatment of Seventh Nerve Paralysis, Ann. Otol., Rhin. & Laryng. **49**:199, 1940.

55. Morwitz, S. M.: Facial Paralysis Associated with Acute Otitis Media, Ann. Otol., Rhin. & Laryng. **37**:1263, 1928.

56. Lillie, H. I.: The Diagnostic Significance of Partial Paralysis of the Facial Nerve in Chronic Suppurative Otitis Media and Mastoiditis, Ann. Otol., Rhin. & Laryng. **50**:38, 1941.

57. Pollak, E.: Die Erkrankungen des facialis, in Alexander, G., and Marburg, O.: Handbuch der Neurologie des Ohres, Berlin, Urban & Schwarzenberg, 1924, vol. 2, pt. 1, pp. 1-76.

tomy in 37, radical mastoid operation in 1 and labyrinthectomy in 3. These operations brought out the following pathologic information: In the 9 cases in which treatment was by myringotomy the otitis subsided and the paralysis disappeared. In 7 of the 37 cases in which mastoidectomy was done for the sole reason that a facial palsy had developed in the patient no sign of osteitis was found. Such an observation was earlier pointed out by Danelius,[58] and Björk [53] has seen similar cases. When further, it is remembered in how many cases the operation reveals enormous areas of osteitis without the patient showing the slightest sign of facial palsy, one must agree with Lund,[59] who said: "The facial palsy seen in acute otitis is no symptom at all of osteitis of the mastoid. In the great majority of cases the paralysis is of toxic origin, even if osteitis of the mastoid exists side by side with it, which, admittedly, is often the case."

The frequency of preoperative facial palsy in cases of acute otitis media is about 0.5 per cent, as earlier shown (table 3). This is exactly the same figure Vogt [60] reported.

Dividing the 50 treated patients according to their age, one finds 21 under the age of 10 years, 11 between the ages of 10 and 20 years and 18 over the age of 20 years. These figures have, of course, only an extremely limited value as I am unable to show how many patients with acute otitis media in the various age groups did not have facial palsy. According to Pollak,[57] young patients, especially children, are particularly inclined to have facial palsy in connection with acute otitis media, and my figures confirm this.

A study of the duration of the otitis prior to the appearance of paralysis has shown that facial palsy may arise at any stage.

It has been the first symptom of otitis in 2 cases. On the whole, however, the older the patients the later it seems to appear. This might possibly find its explanation in the fact that the canal of the facial nerve in the very young person is more thin walled than that in the older person, sometimes even presenting physiologic dehiscences, and consequently more readily permeable for toxins and less resistant toward osteitic processes.

---

58. Danelius, B. D.: Die Fazialislähmung als Indikation für die Aufmeiselung von Processus mastoideus bei akuter Mittelohrsuppuration, Acta oto-laryng. **2**: 355, 1920.

59. Lund, R.: Die Facialisparese bei suppurativen Mittelohrleiden mit besonderem Hinblick auf ihre Verbindung mit labyrinthären Komplikationen und ihre Bedeutung als Operationsindikation. Die postoperative Facialisparese, Ztschr. f. Hals-, Nasen- u. Ohrenh. **23**:296, 1929.

60. Vogt, H.: Die Paralyse des Nervus facialis im Anschlusse an Otitis media acuta, Inaug. Dissert., Heidelberg, 1899; cited by Pollak.[57]

Placement of Facial Palsy in the Syndrome, Especially as an Indication for Operation.—(*a*) Mastoidectomy: Fowler Jr. [43b] wrote: "When a facial paralysis occurs, in association with acute otitis media, mastoiditis is almost invariably present, and as is well known, immediate operation on the mastoid is usually indicated. Rarely, the acute paralysis occurs at a very early stage; then myringotomy may relieve the pressure in the mastoid sufficiently to affect relief of the facial paralysis." Canuyt [61] also regarded the facial palsy in acute otitis media as an indication to perform mastoidectomy in most, though not all, cases. This view I cannot share: First, facial palsy occurring at an early stage is by no means rare; second, it is, as mentioned earlier, of toxic origin and in no way a symptom of mastoiditis. Therefore, I can unconditionally subscribe to Lund's [59] view, which is also shared by others (Baranger [62]; Björk [53]; Manasse [63]; Morwitz [55]; Persky [51] Sirelius [64]): Facial paralysis in association with acute otitis media does not in itself indicate mastoidectomy. Only if the facial palsy is steadily increasing or at least not decreasing after some time of observation and in spite of free drainage through the drum membrane, mastoidectomy is indicated. When other considerations speak in favor of mastoidectomy, facial palsy will of course justify operation.

If, however, facial palsy appears at a late stage, it does indicate mastoidectomy, which, as a rule, will be indicated by other factors. Björk [53] and Dintenfass [65] have expressed the same view.

(*b*) Radical Mastoidectomy: This is an operation which will extremely seldom be considered in cases of acute otitis media (excepting cases in which it is preliminary to labyrinthectomy). In the cases of facial palsy in my material in connection with acute otitis media it has been carried out once, and that only because the osteitic process did virtually represent a kind of spontaneous radical operation.

(*c*) Labyrinthectomy: Facial palsy in association with labyrinthitis has been thoroughly dealt with by Lund [59] on the basis of the same kind of material (though only up to 1928) that is the object of my investigations. So I shall content myself with quoting Lund's con-

61. Canuyt, G.: Fait clinique sur un cas d'otite moyenne aiguë suppurée gauche avec mastoïdite et paralysie faciale périphérique gauche d'origine otogène. Trépanation mastoïdienne. Guérison, Oto-rhino-laryng. internat. **6**:260, 1922.

62. Baranger, A.: Du pronostic et des indications opératoires apportés par la paralysie faciale périphérique au cours d'unne otite myoenne purulente aiguë, Ann. d. mal. de l'oreille, du larynx **45**:1024, 1926.

63. Manasse, P.: Fazialislähmung bei akuter Otitis media, Deutsche med. Wchnschr. **52**:532, 1926.

64. Sirelius, U.: Facial Palsy as Indication for Trepanation in Acute Otitis, Duodecim **51**:329, 1935.

65. Dintenfass, H.: Facial Paralysis from Acute Middle Ear Suppuration, Pennsylvania M. J. **38**:854, 1935.

clusion: "The facial palsy does not indicate labyrinthectomy. The decisive factor is solely the cell count in the cerebrospinal fluid."

Prognosis and Therapy.—In this section the results achieved by conservative treatment of preoperative facial palsy in association with acute otitis media will be surveyed, and on the basis of the reexaminations carried out an attempt will be made at clearing up the question whether better results as regards facial function might have been possible had surgical steps been taken to relieve the nerve.

(*a*) Acute Suppurative Otitis Media with Facial Palsy: In 10 of the cases reexamined every vestige of the palsy had disappeared; in 3 the only remainder was slight synkinesis, and in 13 from a practical point of view the palsy had completely disappeared.

Six of the 9 cases in which myringotomy had been the only treatment were reexamined. In 5 of these it was impossible to demonstrate

TABLE 9.—*Reexamined Cases of Acute Osteitis of the Mastoid with Facial Palsy*

Cases of Facial Paralysis	Degree of Paralysis	Observations on Reexamination
6	2 complete, 4 partial	No vestige of paralysis left
9	6 complete, 3 partial	Mouth was drooping slightly on affected side; in 5 cases mouth slightly less mobile; eye could be closed; Bell's phenomenon was absent; forehead could be wrinkled; all showed synkinesis
1	1 complete	Mouth, eye and forehead were natural; only synkinesis was found
2	2 complete	All three branches were completely paralyzed
18	11 complete, 7 partial	Practically, the result was acceptable in 16 cases and bad in 2

the slightest vestige of the paralysis, and in the sixth the only remainder to be found was very slight synkinesis; i. e., the paralysis had practically disappeared in all 6 cases. Further, in 7 cases simple mastoidectomy had been performed, erroneously on account of the facial palsy, the operation revealing not even the least sign of osteitis. These cases, therefore, belong in this group too. In 5 of these 7 cases the paralysis had completely disappeared, and in 2 feeble synkinesis existed; in other words, in all 13 of these reexamined cases of acute suppurative otitis media with facial palsy the latter had practically disappeared; thus, just as facial palsy in connection with acute otitis media does not indicate mastoidectomy for osteitis, in these cases there was no reason for mastoidectomy, let alone decompression, to secure the future function of the nerve.

(*b*) Acute Osteitis of the Mastoid with Facial Palsy: Eighteen of the 30 cases in which mastoidectomy was performed and osteitis of the mastoid demonstrated were reexamined. The observations are given in table 9.

I shall describe in more detail the 2 cases in table 9 in which the final result was permanent complete paralysis:

The patient in the first case was a boy aged 9 months. For fourteen days before admission he "had a cold"; in the last two days complete palsy appeared on the right side; there was no discharge from the ears. Both tympanic membranes were very red, swollen and bulging. Myringotomy was performed with evacuation of pus. Fifteen days after admission there was still copious discharge of pus, with granulations. Mastoidectomy was carried out on both sides. Six weeks later there was no discharge from the ears, but the palsy was still complete. Two years later the configuration of the mouth in repose was quite acceptable, but there was complete immobility. The eye could not be closed nor the forehead wrinkled, and strong synkinesis was in evidence.

The reason for this bad outcome has without doubt to be looked for in the fact that operation had been postponed too long. In spite of continuous abundant purulent discharge, during which the palsy showed no signs of subsiding, he was not operated on till the twenty-ninth day of illness (the otitis being probably of still longer standing, considering that on admission he had had "a cold" for fourteen days and that myringotomy revealed the presence of thick pus).

The patient in the second case was a man aged 39. Nine days before admission he experienced sudden facial palsy without preceding pain. Only four days later pain was present in and behind the right ear. On admission he showed complete facial palsy and a red, swollen tympanic membrane. Two days later there was infiltration behind the ear with tenderness. Mastoidectomy was performed but showed nothing of special interest except acute osteitis. Reexamination thirty-two years later showed that the mouth was lagging behind considerably on the side which had been operated on and that the ocular slit on that side was wider. The eye could not be closed; Bell's phenomenon was present; the forehead could not be wrinkled; strong synkinesis was noted; faradic irritability was reduced.

In this case at least there was no reason for believing wrong treatment to have been the cause, and yet the toxic damage to the nerve had been so severe that permanent serious disturbance of function persisted.

Table 9 shows that in 7 cases the paralysis had disappeared without leaving the slightest trace, while in 9 cases the remnants of the palsy were so inconsiderable as to be of no practical significance; i. e., a satisfactory result as regards function was observed in 16 of 18 cases. It will be noted further that some remainder of the palsy was demonstrated both in the cases in which it had been complete from the beginning and in those in which it had been only partial; on the other hand, it appears that both complete and partial palsy may disappear without leaving the slightest trace. From this it is obvious that it is impossible to predict a more favorable outcome in the case of partial than in the case of complete paralysis.

It would seem justified to conclude that in the large majority of cases in which facial palsy is encountered with acute mastoiditis it disappears as seen from a practical point of view if only mastoidectomy is undertaken. Simultaneous operations on the canal of the facial nerve (decompression) are not indicated. There are, however, cer-

tain cases in which the palsy does not disappear in spite of mastoidectomy, and these ought to be followed closely. Possibly the faradic test might provide a hint, so that decompression may be performed if response is absent; in this respect I agree with Fowler Jr.[43b]

(*c*) Acute Diffuse Purulent Labyrinthitis with Facial Palsy: Two of the 3 patients suffering from acute labyrinthitis died of meningitis in connection with operation. One is still living and shows no trace of the partial palsy present during his illness.

Chronic Suppurative Otitis Media.

Pathogenesis.—Facial palsy in association with chronic otitis media, especially with acute exacerbation of this condition, may occur as a result of toxic neuritis or inflammatory edema, as does that with acute otitis, but as a rule the nerve is involved in granulating inflammation originating from an osteitic process in the wall of the canal of the facial nerve. As stressed in connection with table 8, the cholesteatomatous variety of chronic otitis media is by far the most frequent cause of facial palsy.

The matrix of the cholesteatoma is located in the epitympanum, more especially at the entrance of the aditus. Beneath the matrix in this narrow space there is granulating osteitis, which together with the pressure by the cholesteatoma makes it possible for the process to spread both into the canal of the facial nerve and into the labyrinth. Vermes[66] described some cases of this type, in one of which postmortem examination revealed a pathologic hole leading into the canal of the facial nerve just above the oval window. Through this aperture the nerve had prolapsed. Kramer,[67] Brooke[20] and Ruttin[67] have described similar cases. Through prolapse of this type the nerve becomes the victim of granulating inflammation. In the causation of such prolapse an important role is played partly by the physiologic dehiscences, partly by the breaking down of the bony canal as a result of the osteitis and partly by the rise of pressure in the nerve canal on account of inflammatory edema.

Numerous cases are encountered, however, in which during operation the nerve is found to be denuded without the slightest sign of facial palsy developing either sooner or later; in Vermes'[66] opinion the reason for this is that the inflammation has not yet reached the perineural sheath of connective tissue. In other cases partial palsy develops in spite of the greatest caution shown in removing cholesteatomatous masses and granulations. Pollak's[57] explanation of this is that the gradually increasing compression of the exposed nerve has resulted in destruction of part of the neurofibrils and injury of the rest and that the gradual rise in pressure allows the latter to adapt themselves to the new condition, so that their function is not impaired to any noteworthy degree. If in a case of this kind the removal of cholesteatomatous masses results in a sudden fall of pressure, the explanation of facial palsy must be that the nerve fibers damaged by the preexist-

66. Vermes, E.: Zur pathologischen Anatomie der otogenen Facialislähmung. Ztschr. f. Hals-, Nasen- u. Ohrenh. **29**:290, 1931.

67. Cited by Fremel, F.: Zur Pathologie der otogenen Gesichtslähmung. Monatschr. f. Ohrenh. **65**:950, 1931.

ing pressure have been unable to adapt themselves to the sudden change, the consequence being that they "collapse."

It is no rare occurrence that only one of the three peripheral branches of the facial nerve is paralyzed. According to Hofmann,[46] this is explained by the fact that within the main trunk the three branches lie separated, each surrounded by strong connective tissue. The branch nearest the site of destruction is involved first, the others remaining intact as long as their sheaths have not been penetrated. According to Canuyt [61] the facial nerve trunk consists of two bundles of fibers, a superficial one conveying impulses to the lower facial muscles and a central one supplying the upper facial muscles. The former is much more exposed to injury on account of its superficial position, whereas the latter will regenerate more easily. In my own material I have a case in which the curet had damaged solely the branch to the oral muscles, while the others had been spared.

My material includes 55 cases in this group, as shown in table 3. The following operations were performed: a radical mastoid operation in 29 cases, labyrinthectomy in 19 and mastoidectomy in 3. In 4 cases

TABLE 10.—*Fifty-Five Cases of Fistula on the Canal of the Facial Nerve*

	Cases of Facial Palsy							
	Preoperative				Postoperative			
	ff	fl	cp	pp	ff	fl	cp	pp
Acute exacerbation in chronic cholesteatomatous otitis media	25	10	23	2	22	14	18	4
Chronic cholesteatomatous otitis media	1	1	1	0	1	0	1	0
Chronic simple otitis media	1	0	1	0	5	1	4	1
Total	27	11	25	2	28	15	23	5

ff = fistula on canal, fl = coexisting fistula on lateral semicircular canal, cp = complete paralysis, pp = partial paralysis.

operation was not resorted to, either on account of the hopelessness of the case or on account of the patients' refusal. The operations yielded the following pathologic information.

In all cases chronic osteitis of the mastoid was demonstrated, and this was of a cholesteatomatous nature in no less than 48 of the 55 cases, the acute exacerbation of the chronic condition playing a particularly predominant role in the genesis of the paralysis. In 27, or one half, of these cases a fistula on the canal of the facial nerve was demonstrated (table 10). In 26 cases this fistula was located on the apex of the facial ridge and in only 1 case in the posterior wall of the external auditory meatus.

Table 10 shows further that together with a fistula on the facial canal there was one on the lateral semicircular canal in somewhat under half of the cases of preoperative palsy and in somewhat over half of the cases of postoperative palsy. The paralysis was nearly always complete. On the other hand, a large fistula may be present on the canal

of the facial nerve without producing the slightest suggestion of paralysis. This observation will be dealt with in detail later.

Position of Preoperative Facial Palsy in the Syndrome, Especially as an Indication for Operation.—While facial palsy in connection with acute otitis media does not in itself indicate operation, the situation is quite different in the case of chronic otitis media. This is explained by the fact that, as mentioned, facial palsy in acute otitis media is as a rule due to edema or to toxic neuritis, seldom to propagating osteitis, while in chronic otitis media just the opposite is the case. Here facial paralysis is due usually to granulating inflammation, and as it is

TABLE 11.—*The Prognosis in Twenty-Seven Cases of Conservatively Treated Preoperative Facial Palsy in Connection with Chronic Suppurative Otitis Media*

Cases of Palsy in Subgroup	Number with Fistula on Canal of Facial Nerve	Degree of Paralysis		Results of Reexamination		
(1) 8	8	Complete	Mouth and forehead immobile	Eye could close	4	In all cases synkinesis was demonstrated
				Eye not closed	4	
(2) 3	1	Partial		Eye could close	3	
(3) 5	4	Complete	Mouth less mobile	Eye could close	5	
				Forehead immobile	2	
				Forehead partly mobile	2	
				Forehead mobile	1	
(4) 4	0	Partial		Eye could close	4	
				Forehead mobile	4	
(5) 2	1	Complete	Mouth, eye and forehead mobile	Synkinesis sole symptom on left	4	
(6) 5 — 3	1	Complete				
(6) 5 — 2	0	Partial		No trace on left	3	
Total number	18 cases of complete palsy, in 14 of which there was a fistula on the canal					
	9 cases of partial palsy, in 1 of which there was a fistula on the canal					

impossible to differentiate in advance the few cases in which the facial palsy is of toxic-edematous origin, facial paralysis in connection with chronic suppurative otitis media presents an absolute indication for radical mastoid operation. This is agreed on in the literature (Canuyt [61]; Lund [59]; Persky [51]). In cases in which the labyrinth is involved, however, facial palsy does not indicate labyrinthectomy; here the cell count in the spinal fluid alone is decisive (Lund [59]).

Prognosis and Therapy.—Since facial palsy following chronic otitis media is, as earlier mentioned, most often due to direct spread of granulating inflammation, causing destruction of the nerve, its prognosis is far more serious than that of facial palsy associated with acute otitis media, of which toxic-edematous damage was seen to be the cause. This was shown to hold good in 27 cases reexamined in this group (table 11).

A closer analysis of table 11 shows with respect to subgroups 1 to 6 certain points of interest.

1. While in this subgroup the mouth and forehead were in all cases completely immobile, 4 patients were able to close the eye, and 3 of these 4 could screw it up tightly. Closing the eye can be effected by relaxation of the levator palpebrae muscle, but for screwing up the eye function of the facial nerve is necessary. Thus one may encounter isolated regeneration of the branch to the eye while at the same time the branches to the mouth and forehead do not function, if, as Fowler Jr.[48b] said, it is not due to "increased education of some other nerve, probably of the IIIrd." The taste was equal on both sides in 2 patients and reduced on the affected side in 1 patient in each of the two groups who could close and not close the eye, respectively. In 2 patients it was not tested.

2. In all the cases of this subgroup the palsy was partial before operation and increased gradually afterward until it became complete. The eye could be screwed up in all 3 cases; i. e., evidence of isolated function of the branch to the eye is met with again.

3 and 4. Here in 1 case the forehead was completely immobile and the mouth showed reduced mobility; it is rare that the frontal muscles function better than those of the mouth.

6. Complete paralysis may disappear without leaving a single trace, not even in the form of synkinesis.

1-6. The position of the mouth in repose in the various subgroups may be stated as follows:

	Both Sides Equal	Affected Side Drooping
(1).....	3	5
(2).....	1	2
(3).....	1	4
(4).....	0	4
(5).....	2	0
(6).....	5	0
Total.....	12	15

The tabulation shows that the mouth may look symmetric at rest even if it is completely immobile.

1-6. Taste was as follows: equal on both sides of the tongue in 20 cases and weakened on the affected side in 4; in 3 cases it was not tested.

Of the 15 cases with fistula on the facial nerve, 13 showed reduced function of the oral branch (with mouth completely immobilized in 8 and partly immobile in 5), 1 showed nothing but synkinesis and 1 had no trace of the palsy left.

Table 11 makes it clear that the prognosis of preoperative palsy in chronic otitis media is anything but good when the treatment is conservative as regards the nerve. If one takes a lenient view of the result and excludes not only the 5 cases in which the palsy had disappeared completely and the 2 in which synkinesis was the only trace left, as well as the 4 with partial palsy in which reexamination showed the mouth to be only slightly less mobile on the paralyzed side, and if

one regards these 16 cases as satisfactory from a clinical and practical point of view, there still remain 11, or almost half of the 27 cases, in which the mouth was completely immobile and 4 cases in which the eye could not be closed.

Are there any clinical clues to the prognosis of the preoperative facial paralysis in association with chronic otitis media? Which measures are to be taken in each case?

Our prognostic considerations might seek support in (*a*) the presence or absence of a fistula on the facial canal, (*b*) the degree of paralysis at the time of operation and (*c*) the result of the faradic test.

It would be tempting to presume that the cases in which operation revealed a fistula on the facial canal had the gravest prognosis. This fistula is nearly always situated at the apex of the facial ridge where the nerve has been eroded by the pressure of osteitis plus cholesteatoma in the epitympanic space at the aditus. It is, however, impossible to distinguish whether the osteitis has affected only the perineural sheath, making this the seat of granulations, or has penetrated more deeply into the nerve. A fistula on the facial canal was demonstrated in 15 cases (table 11). In 1 of these all symptoms disappeared, in 1 synkinesis was shown to be the sole remainder of the former paralysis, and in 5 cases the mouth was somewhat less mobile on the paralyzed side; i. e., there were 7 cases in which the result was considered clinically satisfactory. There still remain 8 cases in which the result was bad, probably because the destructive process had penetrated more deeply into the nerve than in the other 7 cases, a condition which, however, could not be seen at the time of operation. Thus the possible presence or absence of a fistula on the facial canal provides no prognostic indication.

As to drawing prognostic conclusions from the degree of paralysis at the time of operation, this likewise would not be practicable, the result being bad in half the cases of complete and half those of partial paralysis.

Should one contemplate the use of the faradic response as an indicator? The faradic response was not tested before operation in my cases, hence I cannot answer this question with even a fair degree of certainty. Since, however, in half my cases the paralysis was partial and the faradic response therefore presumably positive, even if in a greatly lowered degree, and since the final result in half the cases of partial paralysis, i. e., one fourth of the total number in this group, was nevertheless bad, it is obvious that the faradic test cannot be a reliable indicator. It is possible that a completely negative test may provide a hint; the fact that the faradic excitability is only reduced does not permit any prognostic conclusions.

How, then, should one act when faced with preoperative facial paralysis in connection with chronic otitis media?

If the operation reveals no fistula on the facial canal and no osteitis in the wall, the paralysis may be considered due to toxic damage or inflammatory edema. This was true in 12 of 27 reexamined cases. In 9 of these the paralysis disappeared completely or partly; in 3 (25 per cent) it persisted. The faradic response in these cases is not known. As in my opinion there exist at present no rational means of distinguishing the prognostically good cases from the bad ones, it seems justified in the course of radical mastoidectomy to decompress the nerve and slit open its sheath to relieve the pressure, the more so as this can be done easily and without risk, even if taking this line will lead to decompressive operation in cases in which the paralysis would eventually have disappeared spontaneously. It must, however, not be forgotten that in 3 of the 9 cases characterized as satisfactory the mouth was somewhat less mobile on the defective side. Persky,[51] however, wrote:

> . . . Careful pathologic study can furnish an index of the extent of operative procedure necessary, i. e., as to whether one is to be satisfied with simple exenteration of the diseased cells, with exposure of the facial nerve by the removal of the plate of bone or, finally, with splitting of the sheath of the facial nerve (if edema of the nerve is suspected). But I do not think this assertion is tenable.

In 12 of my cases no macroscopic signs of pathologic processes were to be found after evacuation of the diseased tissue in or around the canal of the facial nerve, and yet the paralysis persisted in 3 cases. So it is clearly impossible to make a good prognosis simply because pathologic processes around the canal of the facial nerve cannot be seen macroscopically.

If operation discloses a fistula on the facial canal, the cause of the palsy may well be toxic-edematous damage but more probably is granulating inflammation that has injured the nerve to a more or less serious extent and possibly eroded it completely. In such a case I am inclined to consider it expedient to inspect the nerve closely, carefully free it of granulations, open its canal to evacuate the osteitis which has crept through the fistula into the canal and attacked the inside, and relieve the pressure undoubtedly exerted by the imflammatory products inside the canal, and finally, should the nerve be completely destroyed, undertake transplantation or suture, if necessary, after the method of Bunnell.[19] I have recently operated in such a case with an 8 mm. long fistula on the canal, which apart from the place where the fistula was located looked perfectly natural. On opening the canal I found considerable osteitis on the inside, extending to a point between the lateral semicircular canal and the styfomastoid foramen. When the nerve sheath was slit open, the nerve bulged out considerably, as if suddenly relieved of heavy pressure.

While the course to be taken (already described) in the case of preoperative facial palsy complicating acute otitis media and, as will be shown later, in the case of postoperative facial palsy is quite clear, preoperative facial palsy in connection with chronic otitis media makes one's considerations difficult. If one adopts a passive attitude toward this paralysis, relying on conservative therapy, the result will be bad in about half the cases, as shown in table 11. It is possible that more direct intervention by the aforementioned method may improve the results, but time will have to decide.

It seems to me that Ballance and Duel [2] characterized the position well by pointing out that in these cases radical operation has already been performed, and adding that "under such circumstances it would be folly not to proceed to deal with the damaged nerve at the conclusion of the mastoid operation." One must also subscribe to the words of John Hunter: "A fixed principle fixes the mind, and a doubtful one leaves it no rest."

POSTOPERATIVE PARALYSIS

Mastoidectomy.

Pathogenesis.—Complete paralysis of the facial nerve occurring in immediate association with mastoidectomy is almost always of traumatic origin. As a rule, the nerve has been damaged a little distally to the bend near the horizontal semicircular canal by a curet, a probe or less frequently, a chisel. During exenteration of a large amount of involved bone with osteitis extending especially into the posterior wall of the external auditory meatus, the curet may hit the nerve in its rotating movements, possibly tearing the nerve completely. The lesion by the probe comes about when one intends to probe the aditus ad antrum but by mistake enters a more distal focus of osteitis in the retrofacial cells, where the nerve may lie exposed; on withdrawal of the probe the nerve is torn.

It rarely occurs that the nerve is directly severed; it may happen, however, especially if the pathologic changes are such that the surgeon loses orientation. But during chiseling a bone spicule may become pressed into the nerve. Finally the canal of the facial nerve may suffer fracture.

Complete facial palsy may arise in immediate connection with mastoidectomy without the nerve having been injured as far as can be made out with the naked eye, namely, when it is involuntarily exposed. If the palsy does not occur immediately after operation, but occurs only hours or days later, the possibility of an interruption of continuity may be disregarded and special attention paid to possible edema or osteitis progressing in spite of operation, whichever is most probable according to the time passed.

In my material there were 37 cases in which facial palsy arose after mastoidectomy; in 27 of these it occurred in immediate association with the operation, and in 10, from one to twenty-one days later. The pathogenesis is shown in tables 12 and 13.

In all 37 cases considerable mastoiditis was found, and in numerous cases the history contained a direct note of its being especially pronounced in the posterior wall of the external auditory meatus.

The pathogenesis will now be dealt with in detail:

(*a*) Paralysis Occurring in Immediate Association with Operation: In 2 cases the nerve had been cut just distally to the horizontal semi-

TABLE 12.—*Twenty-Seven Cases in Which Facial Palsy Occurred in Immediate Connection with Mastoidectomy, Complete in Twenty-Six*

	Cases	Degree of Paralysis
Mechanism of lesion definitely known to have been:		
Actual severing of nerve	2	Complete
Damage inflicted by curet or probe	7	Complete
Fracture of posterior wall of external auditory meatus	1	Partial
Exposure merely, apparently without actual lesion	5	Complete
Mechanism of lesion presumed to have been:		
Injury to nerve in posterior wall of external auditory meatus during exenteration of extensively involved bone	12	Complete

TABLE 13.—*Ten Cases in Which Facial Paralysis Occurred from One to Twenty-One Days After Mastoidectomy*

Cause	Cases	Description of Cases
Edema?	5	Partial, transitory palsy, occurred 1 to 5 days after operation
Hemorrhage	2	Partial, later complete transitory palsy, developed 1 day after operation
Progressing osteitis	3	Partial, later complete palsy, the first occurring 7 days after operation, the second 8 days after, and the third 21 days after. In the first case petrositis developed and the patient died; in the second and third labyrinthitis was present and both patients died.

circular canal, and in 7 cases it had been damaged by curet or probe in the same place. As examples of the latter pathogenesis the following extracts from case reports are quoted:

1. A boy of 14 had retroauricular subperiosteal abscess. At the time of admission the disease had lasted for about a fortnight. Mastoidectomy was performed, revealing pus and granulations, especially in the posterior wall of the external meatus, with perforation of the latter. In the bottom of the cavity a hole was seen, filled with granulations and containing fibers taking a vertical course. By probing and exenteration these were split up to a considerable extent. Complete paralysis was present at the conclusion of operation. The fibers had been mistaken for connective tissue belonging to the sternocleidomastoid muscle. Recovery followed.

2. The patient was a man of 66. The duration of illness on admission was two months. Immediate mastoidectomy revealed pus under pressure, enormous areas

of osteitis, specially in focus VI and in the posterior wall of the external meatus. The involved bone was exenterated by rotating the curet. At the conclusion of operation complete facial paralysis was present. The following day there was explosive meningitis. Petrositis was present. Revision revealed soft tissue in the apex of the mastoid process; apparently the facial nerve had been damaged in the descending portion. The patient died of meningitis.

3. The patient was a man of 56. The duration of illness at the time of admission was seven weeks. Mastoidectomy disclosed considerable acute osteitis in the apical part of the mastoid process and large terminal cells full of pus. On the patient's awakening the facial nerve was found to be paralyzed. The cause is supposed to have been probing of the external meatus, as it took a long time to find the deeply situated antrum.

In the next 5 cases facial paralysis occurred after exposure of the nerve but without direct injury as far as could be made out with the naked eye. Such exposure of the nerve often leads to complete but as a rule transitory paralysis, probably because, without noticing, one contuses the nerve during exposure. That exposure does not in itself produce facial paralysis if the nerve is left untouched has been experienced numerous times, e. g., in cases in which radical operation revealed a big fistula on the canal of the facial nerve without the slightest sign of facial paralysis having been present before or, in some instances, after operation.

Finally, in 1 case facial paralysis appeared as a result of fracture through the posterior wall of the external auditory meatus, the only case in which facial palsy was partial. Whether facial paralysis due to fracture will be partial or complete depends on the course of the fracture line; the findings have varied from slight partial palsy on account of concussion or hemorrhage to permanent complete paralysis on account of actual severance.

The pathogenesis in 15 of the 27 cases of paralysis immediately following operation had been definitely ascertained. There still remain 12 cases to be dealt with, and to illustrate the pathogenesis in this group I quote the following case history:

A girl of 13 years had been ill for one month. She was found to have a subperiosteal retroauricular abscess. Mastoidectomy was performed, showing enormous osseous destruction. Immediately below the horizontal semicircular canal the osteitis extended under the labyrinth to a depth of 6 mm. Immediately after operation complete paralysis was present. This patient recovered.

A common feature of these 12 cases is that all the records contained this note: large or enormous osteitis. One is tempted to suspect that the nerve was injured during exenteration of the pathologic material with the curet, since the osteitis was especially pronounced in the posterior wall of the external auditory meatus. All in all it would seem justified to suppose that in 21 of the 27 cases in question (table 12)

the nerve had been injured by the curet or the probe just distally to the horizontal semicircular canal.

(*b*) Paralysis Occurring After an Interval: If facial palsy does not appear in immediate connection with operation, the possibility of direct interruption of the continuity of the facial nerve may be neglected and edema or hemorrhage suspected to be the cause. In 3 cases palsy of the partial type developed one to five days after operation and subsided. One case was noteworthy in that the operation showed the nerve to be exposed whereas paralysis appeared only on the following day, probably as a result of edema, and was partial and transitory.

In 2 cases facial paralysis likewise began on the day following operation, rapidly developed from partial to complete and later subsided. If, however, facial palsy does not disappear rapidly or does not appear till later, it is a danger signal in that it means osteitis is progressing in spite of operation and indicating revision, possibly more radical intervention. Facial palsy appearing late must consequently be regarded as a symptom of the first importance: All 3 patients belonging to this group in my material died of meningitis.

If one were to draw certain conclusions after surveying cases of facial palsy occurring in connection with mastoidectomy, they would be as follows:

Complete facial paralysis immediately following mastoidectomy is nearly always of traumatic origin. The nerve is damaged in its vertical portion shortly below the right angle bend near the horizontal semicircular canal. The utmost caution should be observed while manipulating the curet in this region, and mastoidectomy should never be postponed till the osteitis has extended so far that comprehensive exenteration in the neighborhood of the nerve canal is necessitated. Finally, regarding probing, it should be kept in mind that not every opening in this region is the aditus but may well be an osteitic cavity, a retrofacial focus, in which the nerve may lie naked and consequently exposed to injury.

The palsies occurring later must raise a grave suspicion of an inflammatory process advancing in spite of mastoidectomy and indicating revision.

Prognosis and Therapy.—Of the 37 patients having facial palsy in immediate association with mastoidectomy, 26 have been reexamined. The grouping of the cases is shown in table 14. The course in each of these groups will be dealt with separately.

(*a*) Severance of the Nerve: The 2 patients concerned were reexamined eleven and fifteen years, respectively, after injury, and the results were found to be identical, namely: The mouth drooped and was immobile on the affected side; the ocular slit on the affected side was wider and

could not be closed (lagophthalmos); Bell's phenomenon was present; the forehead could not be moved; the response to the faradic test was negative; there was not the slightest suggestion of synkinesis. The psychic consequences were grave (the patients occupied prominent positions).

In other words, there was total loss of function of the facial nerve on the affected side. What seems to be nearly the most interesting observation was the heavy droop of the mouth with complete absence of synkinesis. These two phenomena coincide only if every connection between the proximal and the distal portion of the nerve has been broken.

(*b*) Nerve Injured by Curet or Probe: All 3 patients in this group showed slight contracture, raising the mouth to a somewhat higher level on the defective side; all 3 showed complete immobility of the mouth on this side. In 1 patient the injury had obviously involved only the oral

TABLE 14.—*Reexamined Cases of Facial Paralysis Following Immediately After Mastoidectomy*

	Cases
Paralysis occurring in immediate association with mastoidectomy:	
(a) Severance of nerve	2
(b) Injury by curet or probe	3
(c) Exposure of nerve, apparently without actual lesion	5
(d) Fracture of posterior wall of external auditory meatus	1
(e) Supposed injury during exenteration in posterior wall of external auditory meatus	10
Paralysis occurring one to five days after mastoidectomy:	
(f) Edema? Hemorrhage?	5

branch, leaving the eye, the forehead and taste quite intact (faradic test not made). In the 2 remaining patients, the eye could not be closed and was continuously watering, the forehead was immobile and taste and faradic excitability were diminished.

In all 3 patients the paralysis was thus seen to have been a serious permanent affliction, producing complete immobility of the affected side of the mouth. It should be noted that even if the palsy is complete from the start, including all three branches, the remaining two may later function perfectly normally, while the function of the third remains absent. This signifies isolated interruption of this bundle in the trunk of the facial nerve together with only slight simultaneous damage of the other branches, with transitory paralysis.

Further, among the patients who were unable to close the eye on the affected side was one who was noted to have a narrower ocular slit on this side. This was due to relaxation of the levator palpebrae muscle, a phenomenon often mistaken for function of the facial nerve.

(*c*) Nerve Supposed to Have Been Damaged by Curet During Exenteration of Diseased Tissue in the Posterior Wall of the External Audi-

tory Meatus: This pathogenesis was suspected in 10 patients. In 3 patients, the mouth was drooping and completely immobile, the eye could not be closed and watered continuously, Bell's phenomenon was present together with strong synkinesis and the forehead was immobile; electric tests were carried out in 1 case, in which faradic excitability was found lowered. In 4 patients the mouth was slightly drooping on the affected side and somewhat less mobile, though not totally immobile; 3 patients could close the eye; the fourth was unable to do this and presented Bell's phenomenon; none of them was able to wrinkle the forehead. All patients exhibited synkinesis and reduced faradic irritability. In the last 3 patients the only vestige of the paralysis left was a reduction but not absence of function in the frontal muscles together with synkinesis.

Lesion of the nerve during exenteration of tissue extensively involved in osteitis located particularly in the posterior wall of the external auditory meatus may, it is seen, involve serious consequences, ranging from complete severance of the nerve to slight transitory damage. Of 10 patients, 4 showed complete loss of function of the facial nerve branch to the mouth, 3 had greatly diminished function, and only 3 had no permanent signs of any consequence.

(*d*) Fracture of the Posterior Wall of the External Auditory Meatus: This occurred in 1 patient, whose palsy was only partial. But reexamination revealed slight contracture and reduced mobility on the affected side. The patient was unable to show his teeth and suffered from tic. The eye was natural, the forehead could be partially wrinkled, taste was intact. There were synkinetic phenomena and diminished faradic irritability.

Twenty-four years after paralysis had set in, clear signs of it were still in evidence. This shows, as in so many other cases, that full recovery cannot be expected on the strength of the palsy being only partial.

(*e*) Exposure of the Nerve Without Apparent Lesion: This pathogenesis was represented in 5 patients. In 3 of them every trace of the palsy had vanished, synkinesis being absent and faradic excitability unaffected. The fourth patient exhibited as the only remainder slight paresis of the angle of the mouth and faint synkinesis, while the eye, the forehead and taste were all normal. In the last of these 5 patients, the angle of the mouth was slightly less mobile and at a somewhat lower level on the defective side, the eye could be closed but not screwed up, the forehead could not be wrinkled, and there was synkinesis. Taste and electric reaction were equal on the two sides.

A mere exposure of the nerve is thus seen to lead to no consequences of a permanent nature, or to quite insignificant ones: Four patients had escaped practically completely, while 1 showed slight paresis

of the affected side of the mouth. In 4 of the 5 patients no degeneration of axons had taken place, and consequently the condition of the appearance of synkinesis was lacking.

If one surveys the results to see what the prognosis is with conservative treatment of complete facial paralysis occurring in immediate association with simple mastoidectomy and pays special attention to the function of the oral branch, the lack of which in particular disfigures and distresses the patient, they are found to be as follows: Of 21 patients, 12 showed complete loss (8) or great reduction (4) of function of the oral branch, 1 slight reduction of function and 8 normal mobility of the mouth. That is to say that complete facial paralysis occurring in immediate connection with simple mastoidectomy has a very grave prognosis, as more than half of the patients get permanent paralysis of their mouths.

Can these bad results be improved by surgical intervention?

About this there can be no doubt whatsoever. The intratemporal operations on the facial nerve long ago proved their value, and in this connection it is worth emphasizing that a successful result is by no means an isolated phenomenon but is almost invariably the consequence. The cause of the paralysis in cases of the type dealt with here may vary from apparently negligible exposure of the nerve to complete severance; the pathogenesis cannot be ascertained without inspection, and therefore explorative decompression is indicated in these cases.

It is well to keep in mind the words of Ballance and Duel[2]: "The question of operation has little to do with how the paralysis was produced. The reason for operation is that the patient has facial palsy."

It is not justified to wait for a doubtful spontaneous cure which may never come, in view of the serious consequences of facial paralysis, the more so as an explorative decompression of the nerve is a simple operation involving no risks. One would certainly not abstain from performing appendectomy in an absolutely certain case of acute appendicitis because some patients recover without operation.

The objection may, however, be raised that if one acts on this indication for performing radical intervention, explorative decompression of the nerve immediately on the appearance of complete facial paralysis following simple mastoidectomy, this will lead to decompression of the nerve in a number of cases in which the operation might have proved unnecessary. This is admitted, but is appendectomy not performed daily the world over on suspicion in cases in which operation discloses no appendicitis but in which operation was nevertheless absolutely indicated because the possibility of appendicitis could not be disregarded? Even these apparently superfluous operations provide one essential advantage: A good prognosis can now be made with certainty, whereas

before inspection of the nerve one would have had to guess. The one fact which really counts is that the patient has complete facial paralysis the cause of which it is one's unavoidable duty to determine. It is possible that, as proposed by Tickle,[8] one may wait for seventy-two hours after the onset of the palsy only to intervene if the faradic response is lost after that space of time, but that must certainly be the time limit.

What is to be done after decompression of the nerve depends entirely on the conditions present: removal of an impressed fragment of bone, apposition of divided nerve stumps after removal of an interposed blood clot, suture, if necessary, after Bunnel's [7] rerouting method, or free nerve transplantation.

The prognosis of this group under conservative treatment is so bad that an improvement of the results by intratemporal surgical intervention is absolutely indicated.

Radical Mastoidectomy or Labyrinthectomy.

Pathogenesis.—Facial paralysis occurring in immediate association with radical mastoidectomy or labyrinthectomy has on the whole the same causes as that following simple mastoidectomy, namely, first and foremost, traumatic injuries, but while in the case of simple mastoidectomy the nerve is nearly always injured below the bend in its vertical course, the site of the damage in the case of radical mastoidectomy or labyrinthectomy is located on the proximal side of this bend, as a rule at the apex of the facial ridge.

It is easy to understand that the latter operations lead to facial palsy more easily than does simple mastoidectomy, first, because the removal of the posterior wall of the external auditory meatus and the shaving down of the ridge necessitate closer proximity to the nerve, as does also the opening of the cochlea and the vestibule, and, second, because radical mastoidectomy-labyrinthectomy, in contrast with simple mastoidectomy, often is performed in cases of fistula on the canal of the facial nerve where it is almost inevitable that the exposed nerve will be touched or injured.

If facial palsy does not occur in immediate connection with the operation but occurs only after an interval, the possibility of a break of continuity may be disregarded and chief attention paid to hemorrhage in the canal of the facial nerve, edema, progessing osteitis or last, but not least, pressure of tamponade. Facial palsy produced by the last cause may, as pointed out by Lund,[59] occur at any time after the operation, from a few days to several years later. The certain criterion that the palsy is due to pressure of tamponade is that it disappears rapidly if the after-treatment of the operational cavity is altered. The pathogenesis is

most readily understood in the case of a fistula or a dehiscence on the canal of the facial nerve, but an aperture of this kind is by no means a necessary precondition. One can imagine bacterial toxins reaching the nerve from the tamponade by penetrating the shaved-down facial ridge.

In my material are included 120 cases of facial paralysis occurring after radical mastoidectomy–labyrinthectomy. In 84 of these it followed immediately on operation; in the remaining 36 it appeared later. The pathogenesis in these groups is shown in tables 15 and 16.

TABLE 15.—*Eighty-Four Cases of Facial Palsy Occurring in Immediate Association with Radical Mastoidectomy or Labyrinthectomy*

	Cases of Paralysis	
	Complete	Partial
Mechanism of lesion known to be:		
(a) Resection of canal of facial nerve	2	0
(b) Injury to nerve at apex of facial ridge:		
During removal of granulations	4	0
During removal of promontory wall	9	0
Exposure by shaving down ridge	11	4
Manipulations on fistulous canal	23	5
(c) Fracture of canal of facial nerve	5	0
(d) Effect of cocaine	2	0
(e) Compression of nerve against cranial base	0	1
Mechanism supposed to be:		
(f) Hemorrhage in canal of facial nerve	3	0
Mechanism unknown:		
(g) ???	14	1
Total	73	11

TABLE 16.—*Thirty-Six Cases of Facial Palsy Occurring in Connection with Radical Mastoidectomy or Labyrinthectomy After an Interval*

Cause of Paralysis	Cases of Paralysis	
	Complete	Partial
(a) Pressure of tamponade	5	10
(b) Hemorrhage in canal of facial nerve?	5	3
(c) Progressing inflammation	6	7
Total	16	20

The types of pathogenesis in cases of paralysis occurring in immediate connection with operation (table 15) will now be dealt with in more detail:

(*a*) Resection of the Canal of the Facial Nerve: This was done in 2 cases. In the first case it was done by mistake, the canal being resected in the belief that the lateral wall of the external meatus was removed, because a probe had been introduced into a focus deep to the nerve canal and not located, as thought, in the aditus ad antrum. In the second case the canal was removed deliberately with the object of making it possible to master deep panpetrositis.

(*b*) Lesion of the Nerve at the Apex of the Facial Ridge: In 4 cases the nerve had been injured during removal of granulations from the ridge, this pathogenesis being exemplified by the following case records:

1. "After the patient woke up, complete facial palsy was manifest, caused by excision of granulations in front of the facial ridge."

2. "Pale granulations were present in the antrum and the tympanic cavity. The tough membranes adhered strongly to the front of the ridge, and their removal produced facial twitchings. Afterward there was complete paralysis."

This possibility calls for the greatest caution during removal of granulations at the apex of the facial ridge, as the nerve may lie naked between these and consequently much exposed to injury.

During opening of the cochlea and the semicircular canals the nerve is exposed to injury. Five case records contained a note like the following one:

"During the chiseling off of the promontory a couple of rapid jerks were observed on the left side of the face. Complete facial palsy followed."

The nerve may be exposed to injury in other ways during labyrinthectomy, as exemplified by the following entries from case records:

1. "A probe was passed under the facial canal without hindrance, whereby a couple of jerks were produced in the region of the lower facial branch. Afterward complete facial palsy developed."

2. "In the environment of the oval window pronounced osteitis was present and was evacuated by means of a curet, the facial nerve being possibly injured by the procedure. Immediately afterward complete paralysis was observed."

The actual cause of the paralysis cannot be elicited from the case records. The nerve may have been torn completely, or it may have been only touched. The result is identical: paralysis. And the very uncertainty decides one's line of action. That later reexamination raised a definite suspicion of what had actually taken place in many of these cases is a different matter.

While during radical mastoidectomy the nerve is most often injured where it crosses the promontory, it may also be damaged in its vertical course, in the place where it is hit in simple mastoidectomy; in a case in my series it was injured in this place by the burr during labyrinthectomy.

In 28 cases a fistula was to be found on the nerve canal at the apex of the ridge, and manipulations there resulted in complete paralysis in 23 of the cases and partial paralysis in 5.

In the section on preoperative paralysis it has already been mentioned how the osteitis finds its way through the fistula into the canal,

which it destroys from the inside, and how after the sheath has been slit open the nerve bulges considerably, as if relieved of pressure.

(*c*) Fracture of the Canal of the Facial Nerve: This type of pathogenesis is exemplified by the following extracts from case records:

1. "In removal of the wall of the lateral semicircular canal a fairly large fragment of bone was split off the facial canal at the front end of the ridge. Complete facial palsy followed."

2. "An enormous cholesteatoma had eroded the whole of the meatus. The latter was fractured during its removal."

3. "It is unlikely that the facial canal was directly hit, but the bony wall surrounding it was fractured at the close of operation, the fragments, however, remaining in situ."

In all 5 of the cases in this group the paralysis was complete.

(*d*) Cocaine Effect on the Nerve: In 2 cases in which the operation was performed under local anesthesia (infiltration of and behind the meatus, the cocaine inside) complete facial palsy occurred in the middle of the operation.

(*e*) Compression of the Nerve Against the Cranial Base: This type of pathogenesis is illustrated in the following extract from a case record:

"Chronic mastoiditis with perisinous abscess was present. On account of fits of shivering the jugular vein was ligated. During operation the ligature on the anterior facial vein slipped, so the incision was elongated into the retromaxillary region, the lower pole of the parotid gland was split, and the interior jugular vein was compressed against the cranial base. After this procedure there was paralysis of the lower branch of the facial nerve. The cause was probably the firm pressure of the tamponade against the cranial base."

Since the paralysis, as will be shown later, has completely vanished, this supposition is most probably correct, and the other obvious possibility, cutting of the branch, can be disregarded.

(*f*) Hemorrhage in the Canal of the Facial Nerve?: In the cases mentioned in the foregoing groups it was possible to demonstrate the genesis of the paralysis with a fair degree of certainty; in the 3 cases to be mentioned now a supposition will have to suffice. In the 3 case records concerned there is a note to the effect that the bone was extremely hard and that considerable force had to be used in chiseling. One might suppose, though by no means prove, that this led to hemorrhage in the canal, impairing the function of the nerve. No sign of fracture was present.

(*g*) Unknown Cause: There remain 15 cases the records of which do not reveal the cause, because the notes are scanty. Undoubtedly these cases fall into the groups just dealt with. In regard to a great part of them some overlooked traumatic injury seems certain to have been the cause, but this cannot be definitely proved now.

The types of pathogenesis in the cases of paralysis occurring after an interval (table 16) will now be dealt with in detail.

(*a*) Pressure of Tamponade: The most frequently demonstrated cause of this type of facial palsy was pressure of tamponade. As earlier mentioned, the certain criterion is that the palsy disappears when the after-treatment of the cavity of the radical mastoidectomy is altered. Included in this group are 15 cases. The palsy developed at two to twenty-one days after operation—in the majority, after about a week. In 9 of the case records it was noted that during sponging in front of the facial ridge twitchings were observed in the face; so it is easy to understand that the packing in these 9 cases may have caused the palsy. That dehiscence on the facial canal is no necessary condition is proved by the remaining 6 cases: In 5 the palsy when it started was partial; in one or two days, however, it developed into complete paralysis.

(*b*) Hemorrhage in the Canal of the Facial Nerve?: Commenting on this type of pathogenesis, Lund [60] said:

> . . . Wir stehen hier einer Hypothese gegenüber, die indessen das Gepräge der Wahrscheinlichkeit trägt. Um eine Blutung als Ursache annehmen zu können muss dieselbe ganz kurze Zeit, d. h. spätestens einige Tage nach dem operativen Eingriff eintreten, und die Parese muss vorübergehend sein, so dass Restitution innerhalb den ersten Paar Wochen nach dem Anfang der Parese eintritt. (We have here before us a hypothesis, which has the stamp of probability. In order for hemorrhage to be accepted as the cause, it must develop very shortly, that is, a few days after the surgical intervention, and the paresis must be temporary, so that restitution results within the first few weeks after onset of the paresis.)

Quite recently I have operated in a case which gave me a somewhat different opinion. A radical mastoidectomy was performed on a woman of 33 years. After the lapse of about six hours there appeared the first signs of facial palsy. On the following morning this was complete. Seventy-two hours later the faradic response was absent, signifying serious damage to the nerve; so I made a decompressive operation after the method of Ballance and Duel.[2] Below the lateral semicircular canal the nerve was compressed by a black hematoma; this was removed and the nerve sheath slit. Four and one half months later function had returned completely. This seems to prove that hemorrhage in the canal of the facial nerve may produce facial palsy. I quite agree with Lund [60] that a precondition of one's regarding hemorrhage as the cause of the palsy must be that the latter occurs in close connection with the operation (I should say by the next day at the latest), but I do not share this writer's view that recovery will always occur in a couple of weeks. The following case, too, seems to indicate something else:

> A man of 44 underwent a radical operation. After the lapse of some hours facial palsy began to develop, becoming complete nine hours after operation.

Seventy-two hours later the faradic response was lacking, and at this time I ought to have undertaken decompression of the nerve. That was my first case, and I had not yet realized the great value of the decompressive operation. I waited in vain for three months. As there was then still nothing to indicate returning function, I decompressed the nerve. Below the lateral semicircular canal the nerve sheath was thickened (organized hematoma?). It was slit open. Not till seven months later did function begin to return and is now, after fifteen months, good as regards all three branches, but the mouth is not moved to a fully normal extent. In my opinion hemorrhage must have been the cause as a break of continuity can be disregarded on the strength of the interval between operation and palsy and the subsequent operation confirmed the absence of a break. Pressure from tamponade is out of the question, as this was disposed of at once, and the paralysis remained complete for months. Progressing osteitis was not present, the wound healing promptly, so that the only causative factor left seems to be hemorrhage. In comparing the two quite analogous cases reported here, it does not seem unreasonable to suppose a similar genesis for both, and in 1 a hematoma was demonstrated.

Thus facial palsy due to hemorrhage in the canal of the facial nerve may in my opinion remain for months without the faintest sign of restitution being shown.

This group consists of 8 cases. In 5 of them the palsy started to develop on the day of operation after an interval; in 3 it was observed only the next morning.

(*c*) Progressing Inflammation: The last and most important cause of this type of facial palsy is a granulating inflammation which, in spite of the first operation, is progressing in the neighborhood of the facial ridge. As Lund [59] pointed out, the diagnosis is easy, as the paralysis increases in intensity at the same time that a more or less abundantly granulating area is to be seen, often bathed in pus, at the apex of the ridge. This condition demands the quickest possible revision if one is not going to risk permanent facial palsy.

This group consists of 13 cases. In 11 of them there occurred at the same time labyrinthine complications, and in the remaining 2, symptoms of petrositis. The whole question of facial paralysis with labyrinthitis has been dealt with so thoroughly by Lund [59] that I shall confine myself to referring to that writer, only quoting his conclusion: While revision as early as possible is indicated in such cases, the facial palsy is no indication for labyrinthectomy, the only decisive factor being the cell count in the cerebrospinal fluid.

Prognosis and Therapy.—Of 120 patients in whom facial palsy developed in connection with radical mastoidectomy or labyrinthectomy, 85 were reexamined, as shown in tables 17 and 18.

Table 17 reveals that resection of the canal of the facial nerve led, as might have been expected, to complete permanent paralysis of all three branches, and as every connection between the proximal and the

distal portion of the nerve was interrupted, synkinesis was also lacking. The absence of synkinesis was observed in 1 more case, a case in which the vestibule had been evacuated from behind with a curet and the nerve consequently completely torn. All 3 cases were characterized by an immobile, heavily drooping mouth, a wide palpebral fissure with eyelids unable to close, Bell's phenomenon, lagophthalmos and immobile frontal muscles. The sense of taste was tested in the 2 first-named cases, and a peculiar thing was that in 1 it was found to be completely normal in

TABLE 17.—*Sixty Cases of Paralysis Occurring in Immediate Association with Radical Mastoidectomy or Labyrinthectomy*

Type of Pathogenesis	Cases
(a) Resection of canal of facial nerve	2
(b) Injury to nerve at apex of facial ridge	36
(c) Fracture of canal of facial nerve	2
(d) Cocaine effect	2
(e) Hemorrhage in canal of facial nerve	2
(f) Removal of polypus in middle ear	1
(g) Unknown cause	15

	Results on Reexamination	Cases	Palsy Caused By	Cases
Group 1	Mouth immobile	27	Resection of canal	2
	Mouth nearly immobile	10	Excision of granulations	1
	Eye closed, not screwed up	16	Removal of wall of labyrinth	5
	Eye not even closed	21	Shaving down of facial ridge	6
	Forehead immobile	35	Manipulations on fistulous canal	12
	Forehead mobile	2	Hemorrhage in canal	2
	Synkinesis present	33	Unknown cause	8
	Synkinesis absent	4	Removal of polypus from middle ear cavity	1
			Total, 37 cases	
Group 2	Mouth slightly less mobile	6		
	Eye closed and screwed up	6	Shaving down of facial ridge	4
	Forehead immobile	4	Fracture of meatus	2
	Forehead mobile	2	Total, 6 cases	
	Synkinesis present	6		
Group 3	All symptoms had disappeared	17	Removal of wall of labyrinth	2
			Shaving down of facial ridge	2
			Manipulations on fistulous canal	4
			Cocaine effect	2
			Compression against cranial base	1
			Unknown cause	1
			Total, 17 cases	

the foremost two thirds of the affected side of the tongue, a phenomenon to which I shall soon return.

Excision of granulations may have very serious consequences, as seen in table 17, and the utmost caution is called for in this procedure.

Removal of the wall of the promontory or of a semicircular canal led to complete paralysis in 5 cases, in 2 of which the condition disappeared. This is easy to understand if one considers that the lesion may vary from a cutting in two to a mere touching of the naked nerve.

With shaving down of the facial ridge the result may vary in the same way, according to the degree of injury; in 6 cases serious permanent paralysis developed, while in 6 other cases only faint vestiges of the former complete paralysis or none at all could be demonstrated.

Manipulations on a fistulous canal have caused complete or nearly complete paralysis in 12 of 16 cases, and again the necessity of exercising the utmost caution during removal of granulations from the middle ear, especially from the apex of the facial ridge, must be emphasized, as the nerve, without one's knowing it in advance, may lie denuded between the granulations, extremely exposed to serious injury. This was seen with regrettable clarity in a case in which a polypus in the middle ear was seized in a fistula of the antrum via the meatus and bitten off. The patient had maximal permanent paralysis, without the slightest connection later between the nerve stumps, as the heavily drooping mouth and the lack of synkinesis proved. Table 17 shows an interesting thing. In 2 cases of facial palsy due to manipulations of a fistulous facial canal, the mouth was completely immobile and the eye could be closed but not screwed up, while the forehead was fully mobile. This is a rare occurrence, and one recalls Canuyt's [61] opinion that the branch to the mouth lies most laterally within the nerve trunk, being consequently more exposed to injury, whereas the branches to the eye and forehead lie better protected. In my 2 cases the explanation might be that fistulous osteitis had in fact involved and destroyed the branch to the mouth and left the others intact.

Two of the cases reexamined are thought to have been due to hemorrhage in the canal of the facial nerve. Both had a bad prognosis. Two cases were due to fracture of the bony part of the external meatus. In such cases the prognosis naturally varies according to the course of the fracture line. Finally, in 2 cases the paralysis was caused by cocaine and, of course, completely disappeared. Liebermann [68] described a case in which chromic acid had been used as a corrosive at the limen antri, the result being permanent complete facial palsy on account of mummification of the bone. In my material is a quite analogous case, and these 2 instances obviously show that chromic acid should under no circumstances be employed for removal of granulations in the ear.

Special mention will be made of the results of the examinations of taste. These tests were carried out in 44 cases, and in no less than 39 taste over the anterior two thirds of the tongue was equal on both sides, despite the presence among these cases of several in which facial nerve function was completely lacking. In 4 cases taste was less intense and in 1 definitely more intense on the paralyzed side, the latter being a case in which excision of granulations at the apex of the facial ridge had led to complete permanent paralysis. Even in a case in which the canal had been resected, taste was of the same intensity on both sides. This seems to indicate that either the nerve fibers conveying

68. Liebermann, T.: Ein Fall von Fazialisparalysis verursacht durch Chromsäureätzung, Deutsche med. Wchnschr. **48**:905, 1922.

impressions of taste have originally taken other paths than through the chorda tympani, or else nerve fibers in such other paths have taken over the function of those through the chorda later, a confirmation of the discovery of Szende [47] mentioned earlier (page 316).

If the paralysis has developed only after an interval (table 18), the possibility of a break of continuity can be disregarded, and the prognosis in this case is far better.

The reexamined patients whose facial palsy was believed to be the result of pressure of tamponade had all recovered; about half of them, however, exhibited faint synkinesis. In 6 cases facial palsy was supposed to be due to hemorrhage, since it developed one half to one day after operation and since its being due to a break of continuity,

TABLE 18.—*Twenty-Five Cases of Paralysis Occurring After an Interval*

Type of Pathogenesis	Cases
(a) Pressure from tamponade	12
(b) Hemorrhage in canal of facial nerve	6
(c) Progressing inflammation	7
Total	25

Results on Reexamination	Cases	Cause of Palsy	Cases
Mouth immobile Eye could be closed but not screwed up Lagophtalmos Forehead immobile Synkinesis	6	Supposed hemorrhage in canal Progressing inflammation	2 4
Mouth less mobile Eye natural Forehead natural Synkinesis	1	Hemorrhage in canal	1
Mouth, eye and forehead natural	8	Pressure of packings	12
Synkinesis	10	Hemorrhage Progressing inflammation	3 3

pressure of tamponade or progressing inflammation could be excluded. Half of the 6 patients recovered, one exhibiting weak and 2 strong vestiges of the palsy. Finally, in 4 cases the palsy was due to progressing inflammation. In 2 cases there was destructive diffuse labyrinthitis; in 2, petrositis. In all 4 cases the facial palsy persisted.

Summing up the results to see what the prognosis is in cases of complete facial paralysis developing in immediate association with radical mastoidectomy and labyrinthectomy, respectively, and especially stressing absence of function in the oral branch of the facial nerve which is the result that disfigures the patient and inhibits him psychically, one finds the following: Of 60 patients with facial palsy occurring in immediate association with radical mastoidectomy or labyrinthectomy, 37 showed complete (27) or nearly complete (10) loss of function of the oral branch, 6 showed slightly diminished function of the oral branch, and 17 showed completely intact function of the oral branch.

In other words, complete facial palsy occurring in immediate association with radical mastoidectomy or labyrinthectomy has a very grave prognosis, the paralysis of the mouth remaining permanently in more than half of these patients. Exactly the same result was encountered in the cases of facial palsy following simple mastoidectomy and the question *Can these bad results not be improved by surgical intervention?* has to be answered in exactly the same way and with exactly the same motivation: *Without doubt.*

In a foregoing paragraph it was seen that complete paralysis produced by removal of the promontory wall may be due to severance of the facial nerve or to mere slight touching of the naked nerve, and it has been described how manipulations of a fistulous facial canal may lead to permanent or temporary palsy, and exactly the same was the case when the ridge was shaved down or the canal fractured. The only certain thing is that the patient has complete facial palsy, and as long as one does not know the exact cause of this, it is an imperative duty to try to find out. That is the reason why decompression and inspection of the nerve are absolutely indicated if a patient has complete facial palsy in immediate connection with radical mastoidectomy or labyrinthectomy. What further is to be done (suture or grafting) depends on the condition found. A fistula on the canal of the facial nerve indicates decompression, as the osteitis has, as earlier described, crept into the canal and consequently the diseased tissue has to be removed. By following this strict indication the surgeon will, as was explained in the section on palsy following simple mastoidectomy, necessarily decompress the nerve in cases in which the palsy would have disappeared without intervention. It is possible that by waiting seventy-two hours, as proposed by Tickle,[8] these superfluous operations can be avoided, confining active surgical intervention to the cases in which the faradic response is lost by then, but action should under no circumstances be postponed longer.

If facial palsy develops after an interval, the prognosis is far more favorable. If the palsy is due to pressure of packings, it will disappear when these are removed. If, however, one suspects hemorrhage in the canal of the facial nerve as the cause of the palsy, the case should be followed closely by faradic testing: If no response is shown, decompression of the nerve should be undertaken immediately. If the palsy is due, finally, to progressing osteitis, revision should be carried out as soon as possible, combined with decompression of the nerve if the faradic response is lost.

The drawings and the translation from the Danish were made by Dr. Harry Stokholm.

34 Slotsgade, Hilleröd, Denmark.

REPORTED RECOVERIES FROM STAPHYLOCOCCIC MENINGITIS, 1893-1941

WARD J. MacNEAL, M.D.

FRANCES C. FRISBEE, B.A.
AND
ANNE BLEVINS, R.N.
NEW YORK

In the preceding paper we [1] attempted to review the most important pioneer observations on meningitis, in particular those reports of meningitis which might, with some degree of probability, be regarded as examples of staphylococcic infection, up to and including the convincing demonstrations of staphylococcic meningitis in 1890 and 1892. In the present paper we continue the review of outstanding contributions to the study of meningitis after 1892, but now with particular attention to the reported recoveries from staphylococcic meningitis. Unfortunately, even in this modern period of medicine one may find published reports of meningitis in which the specific bacteriologic data are inadequately recorded and the nature of the infectious agent can be recognized only with some uncertainty or not at all.

In the critical evaluation of case reports of meningitis one has to recognize that pleocytosis and symptoms of meningeal irritation may exist without actual bacterial infection of the cerebrospinal fluid in meningism accompanying sepsis, in suppurative inflammations of structures adjacent to the meninges and in virus infections of the meninges. One must also consider that contamination of bacteriologic specimens with staphylococci is a very common source of error and confusion in the clinical application of bacteriologic methods. When staphylococcemia is diagnosed solely on the basis of a growth of staphylococci in a liquid medium inoculated with the patient's blood or staphylococcic meningitis is diagnosed on the basis of a similar growth in a liquid medium seeded with spinal fluid, the clinical success in treatment is likely to appear brilliant. For scientific purposes such laboratory evidence cannot be accepted as valid without most careful scrutiny. On this account we

From the Department of Bacteriology, New York Post-Graduate Medical School and Hospital, Columbia University.

1. MacNeal, W. J.; Frisbee, F. C., and Blevins, A.: Staphylococcic Meningitis from Hippocrates to Le Gendre and Beaussenat: A Mortal Disease, Arch. Otolaryng. **37**:199 (Feb.) 1943.

are inclined to place on the doubtful list those brief and casual remarks about recoveries which have sometimes appeared in the impromptu discussions at clinical congresses. However, the available evidence has been presented in such instances.

In 1893 Macewen [2] gave an impetus to the surgical treatment of meningitis by his report of 17 patients with external pachymeningitis complicating aural disease, all of whom recovered after mastoidectomy and drainage of the dural focus, 12 patients with early local leptomeningitis of whom 6 died without operation and 6 recovered following surgical intervention, and 6 patients with diffuse cerebrospinal meningitis of whom 5 were operated on with 1 recovery. Apparently all of these had primarily an infection of the ear. Bacteriologic studies were not recorded.

Luc [3] in 1897 opened the skull and exposed an area of local leptomeningitis on the frontal lobe of the brain, which had appeared as a sequel to an earlier operation on the frontal sinus. The meningitis was controlled, but later an abscess of the brain developed, which was draining at the time of the report. Bacteriologic examination was not recorded.

Lesné [4] reported the case of an infant 6 months old with multiple furuncles, meningitis and invasion of the blood stream by staphylococci, who after exposure to sunlight and occasional drainage of cerebrospinal fluid recovered so that cultures of the spinal fluid and blood gave negative results. However, the infant continued to have furuncles and eventually died of bronchopneumonia following measles contracted while still in the hospital.

A brief, almost casual, mention of recovery from staphylococcic meningitis complicating pneumonia was recorded by Josias and Netter [5] in 1899. Netter said that he had found Staphylococcus aureus in a pure state in 3 cases of meningitis since the start of the Paris epidemic in 1898. In 1 of these the meningitis complicated pneumonia and terminated in recovery.

---

2. Macewen, W.: Pyogenic Infective Diseases of the Brain and Spinal Cord, Glasgow, James Maclehose & Sons, 1893.

3. Luc: Méningite suppurée aiguë traumatique enrayée par l'ouverture hâtive du crâne et le lavage antiseptique de la pia-mère, Arch. internat. de laryng. **10**:131-143 (March-April) 1897.

4. Lesné: Un cas d'infection staphylococcique du sang et du liquide céphalo-rachidien, Rev. mens. d. mal. de l'enf. **16**:281-288 (June) 1898.

5. Josias and Netter: Méningite cérébro-spinale suppurée due au staphylococcus pyogènes aureus. Hémiplégie droite. Herpès labial en rapport avec une altération du ganglion de Gasser correspondant, Bull. et mém. Soc. méd. d. hôp. de Paris **16**:437-443 (May 5) 1899.

In his book on surgical treatment of cerebral diseases, von Bergmann [6] mentioned briefly a patient who had been operated on for meningocele and showed signs of meningitis four days later. Repeated puncture of the operative site relieved the symptoms, and the patient recovered. Culture of the puncture fluid showed Staphylococcus albus.

Poirier [7] reported the case of a man of 32 who fell on Dec. 6, 1900, got to his feet, started upstairs five steps and fell back, to be picked up unconscious and bleeding at the mouth. After observation in the hospital for a week he went home on December 13. On December 14 he came home from work at about midday because of headache and difficulty with vision, and on the next day he was delirious and was again admitted to the hospital. On December 16 a large trephine opening, 6 by 5 cm., was made on each side of the head 6 cm. above the auditory canal. In both places the dura was opened by crucial incision and a reddish, sticky fluid escaped. Drains were inserted. The patient recovered and was presented before the Société de Chirurgie on Jan. 16, 1901. Preoperative lumbar tap yielded a bloody fluid which on culture produced colonies of Staph. aureus and Staph. albus. Poirier concluded that his early operation had forestalled a lethal meningitis.

Barth [8] was evidently the first to perform successful laminectomy for purulent spinal meningitis. A man aged 21 received a knife stab in the back on Nov. 24, 1900; the external wound was 1 cm. in width and was situated at the right side of the ninth thoracic vertebra. About November 30 the wound was enlarged, and about December 4 there was definite evidence of meningitis. Lumbar tap yielded a purulent fluid containing staphylococci. Laminectomy was performed at once, revealing an extradural abscess. The knife stab had made a slit 5 mm. long in the spinal dura at the level of the eighth and ninth thoracic vertebrae and cloudy spinal fluid was seen escaping from this opening, which was enlarged to 3 cm. and packed with iodoform gauze. As there was no improvement, another laminectomy was done on December 6 at the second and third lumbar vertebrae, and rubber drains were inserted into thick pus. The patient survived with some persistent disability of the lower extremities and weakness of bladder control.

A remarkable case of staphylococcic meningitis was observed in the service of Babinski and evidently reported by him in May 1901, at which time recovery was hoped for. The patient was a police inspector

6. von Bergmann, E.: Die chirurgische Behandlung von Hirnkrankheiten, ed. 3, Berlin, A. Hirschwald, 1899.

7. Poirier: Fracture de l'étage antérieur du crâne, méningite consécutive, trépanation double, guérison, Bull. et mém. Soc. de chir. de Paris **27**:17-20 (Jan. 16) 1901.

8. Barth: Operative Behandlung der eitrigen Meningit's (mit Krankenvorstellung), Verhandl. d. deutsch. Gesellsch. f. Chir. **30**:51-54, 1901.

41 years of age. Many spinal taps yielded cloudy fluid containing Staph. aureus. The illness began on Dec. 15, 1900 and ended in death on Aug. 16, 1902. At autopsy there was pus distending the lumbar cul-de-sac and extending upward along the cord to surround the cerebral peduncles. The possibility of antecedent meningococcic infection could neither be confirmed nor excluded. The final report was made by Léri [9] in 1902.

Göbell [10] in 1909 recorded a well established example of recovery from a purulent meningitis due to Staph. aureus, of the primary spinal type, which probably originated in a vertebral osteomyelitis. Laminectomy and spinal drainage were followed by recovery. In the subsequent discussion Kümmell reported a case of cerebrospinal meningitis with positive culture of Staph. albus from the spinal fluid in which recovery followed multiple spinal taps. He evidently regarded staphylococcic infection of the meninges rather lightly, and his case may be regarded as not adequately reported for scientific acceptance.

Preysing [11] in 1912, in an extensive review of the subject at the German Otologic Congress, said that probably every surgeon present had in his own practice induced or at least hastened a meningitis by operation and that the statistics of such unfortunate results, if assembled and displayed, would make the operative recoveries seem few indeed. However, he expressed optimism for the future. Preysing's own case was that of a boy with otitis media and symptoms of meningitis, and with Staph. albus in the cloudy spinal fluid, who recovered after paracentesis and lumbar puncture.

Bourges [12] reported a valid recovery. A seaman fell ill with violent meningeal symptoms on Dec. 10, 1913 and was admitted to the hospital on the next day. Immediate spinal tap yielded a purulent fluid, and meningococcic serum was administered. Examination of the fluid revealed the presence of gram-positive cocci. Repeated spinal taps confirmed the original finding, and Staph. albus was grown in pure culture. Turpentine was injected into the thigh on December 29 to induce a fixation abscess, and incision of this on January 3 yielded 200 cc. of pus containing Staph. albus. Gradual improvement was followed by complete recovery on March 25.

---

9. Léri, A.: Méningite cérébro-spinale à forme ambulatoire ayant évolué pendant huit mois, Arch. gén. de méd. **7**:395-414, 1902.

10. Göbell: Ätiologie und Diagnose der Meningitis spinalis purulenta, Zentralbl. f. Chir. **36**:1179-1180 (Aug. 21) 1909.

11. Preysing, H.: Bericht über die Heilbarkeit der otogenen Meningitis, Verhandl. d. deutsch. otol. Gesellsch. **21**:23-62, 1912.

12. Bourges, H.: Méningite cérébro-spinale suppurée et primitive à staphylocoques purs, sans autre localization purulente, Rev. gén. de clin. et de thérap. **30**:68-72 (Jan. 29) 1916.

The recoveries reported by Niles [13] in 1917, by Rocaz [14] in 1920 and by Schönbauer and Brunner [15] in 1920 are lacking in certain essentials for confident acceptance as examples of staphylococcic meningitis, and they have been placed on our doubtful list. On the other hand, the recovery reported by Lewkowicz [16] in 1920 seems valid. The patient was a soldier who survived a gunshot wound of the cranium, inflicted on Oct. 4, 1916. After healing of the craniotomy wound he evidently had a sluggish abscess of the brain which ruptured into the cerebral ventricle to give rise to meningitis with repeated exacerbations. Lumbar tap yielded a purulent fluid containing Staph. aureus. Staphylococcus vaccine was used by subcutaneous injection, increasing to a dose of 20,000,000,000 organisms. The patient had remained well for two years before the case was reported.

Two survivals reported by Lortat-Jacob and Grivot [17] in 1920 seem well established. In both patients otitis media developed during the puerperium. As soon as signs of meningitis appeared and staphylococci were demonstrated in the cloudy spinal fluid, mastoidectomy was done and vaccine therapy instituted. One patient recovered completely, and the other survived with atrophy of the optic nerve.

Ayer [18] in 1924 mentioned his success in draining the cisterna magna in a case of staphylococcic meningitis. Data were not reported in regard to survival of the patient.

Dandy [19] in 1924, in a paper chiefly concerned with the technic of cisternal drainage previously exploited by Haynes and by Day,[20] mentioned 3 cases of staphylococcic meningitis, apparently instances of recovery, without presenting details. The second case in this report

13. Niles, W. L.: Meningitis: Three Cases; Two of Meningococcic and One of Staphylococcic Origin, M. Clin. North America **1**:745-765 (Nov.) 1917.

14. Rocaz: Méningite à staphylocoques traitée avec succès par les injections intrarachidiennes d'étain colloidal, Gaz. hebd. d. sc. méd. **41**:218-219 (May 9) 1920.

15. Schönbauer, L., and Brunner, H.: Zur Behandlung der Meningitis mit Staphylokokkenvakzine, Wien. klin. Wchnschr. **33**:491-492 (June 3) 1920.

16. Lewkowicz, K.: Guérison par un vaccin spécifique d'un abcès cérébrale et d'une méningite générale à staphylocoques, Arch. de méd. d. enf. **23**:540-545 (Sept.) 1920.

17. Lortat-Jacob, L., and Grivot, M.: Deux cas de méningites généralisées à staphylocoques—Utilité des vaccinations staphylococciques associées à la trépanation et aux rachicentèses. Guérison, Bull. et mém. Soc. méd. d. hôp. de Paris **44**:1555-1559 (Dec. 16) 1920.

18. Ayer, J. B.: A Brief Review of Certain Mechanical Considerations in the Treatment of Meningitis, New York State J. Med. **24**:389-391 (March 21) 1924.

19. Dandy, W. E.: The Treatment of Staphylococcus and Streptococcus Meningitis by Continuous Drainage of the Cisterna Magna, Surg., Gynec. & Obst. **39**: 760-774 (Dec.) 1924.

20. Day, E. W.: Indications for and Results of Operative Treatment of Otitic Meningitis, Surg., Gynec. & Obst. **16**:369-376 (April) 1913.

seems to be a valid instance of recovery. In a boy of 11 years meningitis developed after a lumbar puncture. He recovered in fourteen days and was well five months later. He was treated by suboccipital surgical drainage of the cisterna magna. The specific nature of the bacteria concerned is somewhat uncertain because of confusing conflict between the clinical charts and statements in the text of this paper. The report of Barker [21] also leaves something to be desired in regard to the diagnosis of meningitis and in respect to recovery of the patient, who at last report was confined to bed with a hopeful prognosis. No later report has been found.

Sicard and Paraf [22] reported a well established example of ascending meningeal inflammation which evidently originated in an abscess of the buttock. There was spinal block at the level of the tenth thoracic vertebra. Laminectomy extending from the fifth lumbar to the tenth thoracic vertebra and incision of the dura for a distance of 40 cm. drained a series of small staphylococcic epidural abscesses as well as the subdural space. At the time of the report the patient was able to walk, but there was still a small persistent fistulous tract. The French surgeons have been particularly successful in dealing with this type of staphylococcic spinal meningitis by laminectomy.

Dupérié, Rocher and Auriat [23] also reported a case of this general type. The patient was a boy of 3 with a recognized osteomyelitis of the arc of the second lumbar vertebra and staphylococci in the purulent spinal fluid. The dural sac was incised and drained. Later the posterior arcs of the first and second lumbar vertebrae were completely resected, a large dural incision was made and a drain was inserted beneath the dura for a distance of several centimeters. Colloidal tin and staphylococcus vaccine were given. The patient recovered with slight disability of the left lower extremity but was able to walk.

Another example of success in dealing with this type of meningitis was reported by Garber.[24] The patient was a girl of 14 years who had suffered a severe fall on the sacrum three years before. Her meningitis began in September 1922 with severe lumbosacral pain, headache and high fever. Lumbar tap yielded creamy pus containing many staphylo-

21. Barker, L. F.: Recovery from Staphylococcus Septicaemia with Meningitis, Thrombophlebitis, Embolic Pneumonia and Nephritis, Internat. Clin. **1**:1-13, 1925.

22. Sicard and Paraf: **Épidurite ascendante à staphylocoque. Radio-lipiodol. Laminectomie. Guérison,** Bull. et mém. Soc. méd. d. hôp. de Paris **49**:50-52 (Jan. 16) 1925.

23. Dupérié, R.; Rocher, H. L., and Auriat: **Méningite aiguë à staphylocoques consecutive à une ostéo-myélite d'un arc vertébral. Guérison,** Gaz. hebd. d. sc. méd. **46**:196-197 (March 29) 1925.

24. Garber, F. W.: Staphylococcus Meningitis Limited to the Spinal Canal: Puncture of Cisterna Magna, J. Michigan State M. Soc. **24**:597-599 (Nov.) 1925.

cocci, but cisternal tap yielded a slightly cloudy fluid with 190 cells per cubic millimeter, which was negative on culture. Roentgenologic examination disclosed osteomyelitis involving the right sacroiliac joint. The arch of one lumbar vertebra was removed, and a rubber tube was inserted into the spinal canal for drainage. Pus pockets in the sacrum were also drained. Subsequently the wound was irrigated periodically with hot water. The patient was able to sit up after two months and walked out of the hospital after a year, although some contracture persisted.

A somewhat doubtful example was a case reported by Wharry [25] as one of otogenous staphylococcic meningitis. The patient had a mastoidectomy (right side) on Sept. 1, 1924, tonsillectomy and adenoidectomy on September 22 and incision of the mastoidectomy scar on December 4. On April 19, 1925, when she was 2 years old, she was admitted to the hospital with a diagnosis of meningitis. Spinal tap yielded a "tubeful" of turbid fluid. Bilateral radical mastoidectomy was performed, and a large fistula in the right semicircular canal was discovered. On the next day spinal tap again yielded cloudy fluid. Locke's solution at body temperature was allowed to run through the lumbar needle into the spinal canal with the head lowered so that some of the fluid came out of the mastoidectomy wound. The patient suffered a collapse, but was much improved on the next day and progressed to complete recovery. Cultures of the specimens of spinal fluid failed to show any growth of bacteria, and hence the diagnosis of staphylococcic meningitis lacks confirmation. The use of intrathecal lavage is an interesting feature of the treatment.

Moise [26] obtained a recovery in a case of spinal meningitis arising from a congenital sacrococcygeal sinus, with Staph. albus present in the cloudy spinal fluid. The sinus was excised and laminectomy of the first sacral segment performed, with insertion of a rubber drain beneath the dura. The patient recovered after an illness of two months.

The patient reported by Emerson [27] was a school girl 12 years of age who first complained of pain in the left hip on May 16, 1924. On May 23 her neck became stiff and her legs somewhat spastic. Lumbar tap yielded no fluid, but a cisternal tap brought forth 4 cc. of normal clear fluid. However, on May 24, when a larger needle and suction were employed, a lumbar tap yielded 2 cc. of thick pus which on culture revealed Staph. aureus. After laminectomy of the third and fourth

25. Wharry, H. M.: Three Cases of Middle-Ear Disease with Intracranial Complications, Brit. M. J. **1**:85-87 (Jan. 16) 1926.

26. Moise, T. S.: Staphylococcus Meningitis Secondary to a Congenital Sacral Sinus, Surg., Gynec. & Obst. **42**:394-397 (March) 1926.

27. Emerson, K.: Staphylococcal Infection of the Spinal Meninges, Boston M. & S. J. **196**:465-467 (March 24) 1927.

lumbar vertebrae, the dura was incised and a Penrose drain inserted. This drain was removed after about a week, and then irrigations of diluted solution of sodium hypochlorite (Dakin's solution) were used. The meningitis disappeared. Two months after the operation the right elbow joint became inflamed, and the same organism was found in the drainage. This lesion healed with ankylosis. Later metastatic lesions appeared in the right radius and in the right ilium, and in August 1926 another developed in the left thigh. The author suspected that in this patient the meningitis was secondary to a lumbar vertebral lesion, but careful search by roentgen examination failed to detect it.

A hydrocephalic female infant was treated by ventricular puncture on Feb. 9, 1927, at which time phenolsulfonphthalein was injected. Spinal puncture twenty minutes later yielded bloody fluid. On the next day punctures of the lateral ventricle, the cisterna and the lumbar region all yielded cloudy fluid containing many leukocytes and gram-positive cocci, which on culture proved to be staphylococci. Lamb [28] injected 7 cc. of 0.1 per cent solution of gentian violet into the lateral ventricle on February 20 and again on February 24. There was gradual improvement, and the fluid obtained on March 30 was sterile. The patient was discharged April 14, at which time the circumference of the head was 45.5 cm., a reduction from 47.75 cm. on February 8.

Baranger [29] reported the case of a boy 15 years of age in whom pain and swelling developed over the first lumbar vertebra, extending to the left. A paravertebral incision on July 20, 1927 released pus containing Staph. albus, and a denuded bony lamina was resected. Two days later the neck was stiff, and on July 26 the spinal fluid was purulent. The patient received acriflavine hydrochloride and autogenous vaccine (apparently lysate vaccine of Duchon) intravenously, and later the vaccine and a silver preparation were injected into the spinal canal. Neoarsphenamine, autoserum intrathecally and several transfusions were also given. The boy recovered and returned to school on November 1.

Manicatide, Popovici-Lupa and Constantinesco [30] treated successfully a boy aged 4 years from whom thick pus was obtained by spinal punctures in the middorsal and cervical regions. The cultures revealed Staph. aureus. The patient was treated by intrathecal lavage with 400 to 500 cc. of physiologic solution of sodium chloride at body temperature without untoward reaction and was also given daily subcutaneous

28. Lamb, F. H.: Report of a Case of Staphylococcus Meningitis Treated with Intraventricular Injections of Gentian Violet, Arch. Pediat. **45**:306-309 (May) 1928.

29. Baranger, J.: Deux observations d'ostéomyélites aiguës de la colonne vertébrale, Bull. et mém. Soc. nat. de chir. **54**:789-795 (June 9) 1928.

30. Manicatide, M.; Popovici-Lupa, M., and Constantinesco, C.: Sur la méningite purulente à staphylocoques chez l'enfant, Rev. franç. de pédiat. **4**:633-645 (Oct.) 1928.

injections of autogenous vaccine. The lavage was given repeatedly from Jan. 15 to Jan. 19, 1928. The patient was discharged cured on February 5. Their second patient was a boy aged 5 years who was thrown to the ground by a playmate on April 26, 1928 and remained unconscious for an hour. He then walked home. That evening he had a chill followed by fever and delirium, stiffness of the neck and retention of urine. On May 5 a lumbar tap permitted the aspiration of creamy pus containing staphylococci. On May 6 the first intrathecal lavage was given, the fluid entering between the seventh and eighth thoracic vertebrae and escaping through needles between the second and third and between the fourth and fifth lumbar vertebrae, with the patient sitting upright. Lavage was continued until the outflow, at first purulent, had become completely clear. Daily lavage was continued to May 10 and at greater intervals thereafter. Camphorated oil and autogenous vaccine were given subcutaneously. The patient was discharged from the hospital, entirely recovered, on June 17. The authors thought that the disease was confined to the spinal meninges in both these patients.

Bezançon and his associates [31] reported a somewhat similar case, in which the patient was an adult. A woman aged 30 had a history of miscarriage in December 1926. On April 15, 1927 her illness began with chill, intense headache and vomiting. She was admitted to the hospital on April 23 with all the classic manifestations of meningitis. Lumbar tap yielded a cloudy fluid, negative for bacteria. There was edema of the lumbar region and contracture of the right thigh at this time. Cultures of blood taken on April 26 and April 28 gave a growth of Staph. aureus, and the spinal tap on April 28 yielded a frankly purulent fluid containing the same bacteria. Beginning on April 29, daily injections of a lysate vaccine prepared by Duchon were given intravenously, intrathecally and subcutaneously. The intravenous injections were followed by a shock reaction with severe chill. On May 2 and thereafter the spinal fluid was normal. A fixation abscess was induced in the thigh on June 2 and incised on July 12. The psoas muscle was incised on August 12 and pus drained. At this time roentgenologic examination disclosed spondylitis of the twelfth thoracic and first lumbar vertebrae. In June 1928 the patient was well except for a persistent paravertebral fistula from which staphylococci could still be obtained. Possibly the spondylitis preceded the meningitis in this patient.

The accumulation in the French literature of these successes in dealing with purulent spinal meningitis originating from lumbar osteo-

31. Bezançon, F.; Weismann-Netter, R., and Destouches: Un cas de staphylococcémie avec méningite suppurée ayant abouti à la guérison, Bull. et mém. Soc. méd. d. hôp. de Paris **44**:1764-1768 (Dec. 20) 1928.

myelitis was signalized in 1929 by Chavany and George,[32] who recognized a special form of staphylococcic meningitis, *"La forme rachidienne primitive,"* in which survival may be expected. Their patient who recovered was a woman of 22 who was seized with violent lumbar pain on Feb. 1, 1925. Spinal tap on February 3 yielded normal fluid. Fluid obtained by later taps was cloudy but free from bacteria through February 13. Culture of blood taken on February 14 gave a positive growth of Staph. aureus, and the same organism was found in the spinal fluid of February 18 and in pus aspirated from the lumbar puncture on February 23. Autogenous vaccine was used for a long time. The patient was discharged from the hospital well on May 29.

The thesis of Benoit [33] served to focus attention on meningitis caused by the staphylococcus and to clarify the prognostic differences between the various types of this disease. First, there are the meningeal complications of staphylococcemia, which include (*a*) meningismus, a symptomatic but not a genuine meningitis, (*b*) acute purulent cerebrospinal meningitis, (*c*) subacute purulent cerebrospinal meningitis, (*d*) fulminant cerebrospinal meningitis and (*e*) the serous type of cerebrospinal meningitis. Second, there are the types of staphylococcic meningitis arising by extension from a recognizable focus of inflammation: (*a*) otogenous; (*b*) from furuncle of the face, scalp or neck; (*c*) from an unrecognized or recognized focus in vertebrae, intervertebral disks or adjacent tissues, the *"méningite rachidienne primitive"* of Chavany and George,[32] of which there are malignant and benign varieties; (*d*) the silent type, forme fruste, in which the disease is first suspected at autopsy. Third, there are the post-traumatic forms of staphyloccocic meningitis, in which the inflammation of the meninges may supervene a long time after infliction of the wound. This type has been observed particularly following war wounds. Benoit suggested that the apparently primary meningitis may take its origin from an unrecognized latent focus in bone, just as may be the case with clinical osteomyelitis. He cautioned against ill considered spinal taps in the primary spinal form, because this procedure may carry infection from an epidural purulent collection into the leptomeninges and so transform a fairly benign disease into one with grave prognosis. Benoit added one recovery to the list, that of a boy of 12 years with multiple lesions of osteomyelitis and lumbar suppuration involving the meninges due to Staph. albus, with recovery after surgical drainage and administration of autogenous

32. Chavany, J. A., and George, P.: Sur une forme spéciale de méningite staphylococcique. La forme rachidienne primitive, Presse méd. **37**:862-864 (July 3) 1929.

33. Benoit, M.: Contribution à l'étude de la méningite à staphylocoques, Thesis, Toulouse, Henri Cléder, 1929.

vaccine and methenamine. This same patient has been the subject of a separate report by Laporte, Calazel and Benoit.[34]

Sciaroni [35] reported a similar success with a boy of 16. Thick pus was aspirated from the lumbar portion of the spine, which on culture was found to contain Staph. aureus. Laminectomy of the second lumbar vertebra was performed, with insertion of a drain. Recovery occurred after six weeks.

Ballenger [36] reported on a boy of 6 years in whom meningitis developed as a complication of purulent rhinitis. The spinal fluid contained 320 cells per cubic millimeter, and Staph. aureus was obtained by culture of specimens taken on Aug. 17, 19, 25 and 30 and Sept. 11, 1929. On August 20 an external opening was made into the left frontal air cavity of the cranium and through it into its fellow air cavity and also through the posterior wall to expose a dural abscess. Osteomyelitis of the cranium was recognized. On September 11 complete paralysis of all the right extraocular muscles appeared. The patient improved, and only a small discharging sinus remained on Feb. 21, 1930.

The patient of Lewis,[37] reported as having recovered from thrombosis of the cavernous sinus in 1931, died later (1933), and at autopsy there was evidence of an old infection of the meninges. The case requires mention, although it is not acceptable as an instance of recovery.

Although Bezançon and his associates [31] employed a "lysate vaccine" prepared by Duchon in their successful treatment reported in 1928, the first report of the use of a bacteriophage, designated as such, to bring about recovery from staphylococcic meningitis appears to be that of Stout,[38] a pioneer observation not featured in the title of his publication. The patient was a girl of 16, on whose chin a furuncle developed on July 19, 1930. This was lanced on July 23 and cauterized deeply on July 24. By July 26 the clinical picture of thrombosis of the cavernous sinus was fully developed. Culture of blood taken July 28 gave a growth of Staph. aureus. She was treated with bacteriophage until August 2,

34. Laporte, Calazel and Benoit: Méningite cérébro-spinale purulente aiguë à staphylocoques, suivie de guérison, Bull. et mém. Soc. méd. d. hôp. de Paris **45:** 1367-1370 (Dec. 2) 1929.

35. Sciaroni, G. H.: Surgical Treatment of Staphylococcus Meningitis, California & West. Med. **32:**186 (March) 1930.

36. Ballenger, H. C.: Meningitis (Staphylococcus Aureus) Secondary to Frontal Sinusitis: Operation and Recovery, J. A. M. A. **94:**1040-1043 (April 5) 1930.

37. Lewis, E. R.: Cavernous Sinus Thrombosis: Recovery, Ann. Otol., Rhin. & Laryng. **40:**341-347 (June) 1931; Cavernous Sinus Thrombosis: Recovery Confirmed by Necropsy Three Years Later, Tr. Am. Laryng., Rhin. & Otol. Soc. **40:**378-385, 1934.

38. Stout, B. F.: Septic Cavernous Sinus Thrombosis: Report of Two Cases with Recovery of One Following Bacteriophage Therapy, J. Lab. & Clin. Med. **17:** 28-39 (Oct.) 1931.

when this therapy was discontinued. On August 23 pain in the head and neck and vomiting, together with a rise of temperature to 103.2 F., announced the advent of meningitis. Lumbar tap at 4:00 p. m. yielded 15 cc. of turbid fluid under high pressure with a cell count of 1,100 and numerous staphylococci in the direct smear and on culture. One cubic centimeter of bacteriophage was given intrathecally. At 11:00 a. m. August 24, the spinal fluid showed only 370 cells. Four cubic centimeters of bacteriophage was injected at this time. Doses of 5 cc. were then continued daily for four days, including August 28. Five cubic centimeters was again administered intrathecally on September 3. Cultures of the spinal fluid specimens of August 23 and 25 yielded staphylococci, but the fluid obtained on August 28 did not contain the organisms. The patient recovered and was free from sequelae in December 1930.

An example of epidural abscess in the lumbar region in a girl of 6, with spread of the infection to the leptomeninges by the puncture needle, was reported by Rosamond.[39] Laminectomy and insertion of a drain through the dura were followed by recovery after several months.

Schless,[40] evidently without knowledge of Stout's observation, obtained a good result in a case of staphylococcic meningitis. The patient was a boy of 13 who had a large furuncle some time in June 1931. On July 14 he complained of pain in the back, and by July 18 he was semistuporous, with the classic signs of cerebrospinal meningitis. Spinal tap yielded 20 cc. of turbid fluid containing 6,000 cells per cubic millimeter and no reducing substance for Fehling's solution. On culture it yielded a growth of Staph. aureus. The subsequent taps confirmed these findings. On July 22 (morning), after removal of 30 cc. of turbid fluid, an intrathecal injection of 5 cc. of broth staphylococcus bacteriophage prepared by Swan-Myers Laboratories was given. In the evening another tap was performed, and 10 cc. of the phage was given intrathecally. Spinal taps were continued twice a day, and each time 10 to 15 cc. of phage was injected. Cultures of all specimens of spinal fluid revealed Staph. aureus until the phage was given on July 22, and they all gave negative results thereafter. The patient was discharged from the hospital twenty-three days after admission, completely recovered and without sequelae.

Stout[41] in 1933 wrote again in regard to his first instance of recovery,[38] and reported also another recovery of a patient in whom

39. Rosamond, E.: Epidural Abscess Complicated by Staphylococcic Meningitis: Report of a Case with Complete Recovery Following Operation, J. Pediat. **1**:230-232 (Aug.) 1932.

40. Schless, R. A.: Staphylococcus Aureus Meningitis: Treatment with Specific Bacteriophage, Am. J. Dis. Child. **44**:813-822 (Oct.) 1932.

41. Stout, B. F.: Bacteriophage Therapy, Texas State J. Med. **29**:205-209 (July) 1933.

staphylococcic meningitis developed after spinal anesthesia. The bacteriophage was injected intrathecally twice daily in doses as large as 20 cc. In this paper Stout mentioned that "opinion in high places" is adverse to the therapeutic use of bacteriophage but that good results cannot be explained away.

Bourrel [42] in his thesis mentioned briefly, without reference, a recovery obtained by Iacobovici in a patient in whom staphylococcic meningitis developed nine days after fracture of the skull. Daily intrathecal injections of antistaphylococcus serum, methenamine and electrargol were given along with large doses of serum subcutaneously and intramuscularly.

Arauz and Tato [43] treated an otogenous streptococcic meningitis with streptococcus serum and a preparation of cinchophen and methenamine after mastoidectomy. The patient had a relapse on the twenty-fifth postoperative day, and both streptococci and staphylococci were found in the spinal fluid. The mastoidectomy wound was revised and the posterior fossa explored. The preparation of cinchophen and methenamine was again given intrathecally, and the patient became symptom-free after four days. The evidence of staphylococcic meningitis would appear to be somewhat uncertain in this case.

A woman aged 28 who had been shot in the head with a revolver at short range was completely paralyzed but conscious. Lumbar tap yielded bloody fluid. After eight days the entrance wound had healed. Total blindness remained. On the ninth day stiff neck, severe headache, vomiting and obstinate constipation developed, accompanied with a positive Kernig sign and a temperature of 39.8 C. (103.6 F.). Barthélemy [44] then saw her for the first time. Lumbar tap released cloudy fluid under high pressure, containing abundant leukocytes and staphylococci. A second specimen, removed two days later, was still more cloudy. At this time one ampule of *bactéstaphyphage* was injected into the spinal canal. Without other treatment the patient improved and was out of bed in eight days. Two months later she was in excellent condition except for persistent blindness. The bullet was located roentgenologically near the sella turcica. It seemed to be well tolerated.

Wounds of the skull have been among the important factors causing death in war. Persons with such wounds who have survived to reach the hospitals at the rear have in large measure succumbed to meningitis

42. Bourrel, M.: Étude du lavage ventriculo-spinal à propos de trois cas de méningites post-traumatiques, Thesis, Toulouse, J. Castellir, 1934.

43. Arauz, S. L., and Tato, J. M.: Meningitis otógena a estreptococo. Operación. Curación. Recidiva meníngea a estafilococo. Revisión operatoria. Curación, Rev. Asoc. méd. argent. **49**:198-201 (Feb.) 1935.

44. Barthélemy: Méningite traumatique à staphylocoques. Injection intrarachidienne de bactéstaphyphages. Guérison, Bull. et mém. Soc. nat. de chir. **61**: 321-322 (March 9) 1935.

in which streptococci and staphylococci have played the chief role. Dmitriev [45] estimated that deaths from gunshot wounds of the cranium in the Russian hospitals from 1914 to 1920 were largely due to meningitis. As high as 97 per cent of the deaths in one series to which he gave personal attention were ascribed to this cause. Infection with staphylococci seemed to be especially malignant.

Dunlap [46] reported the case of a boy of 8 who hurt his knee and his back at play on Sept. 27, 1934. On October 1 he was admitted to the hospital with a rigid neck, a temperature of 101 F. and a tender cyst over the lumbar spine. Spinal tap yielded a cloudy fluid under high pressure, with gram-positive cocci in the smear and pure Staph. aureus on culture. At the time of the first puncture meningococcus serum was injected into the spinal canal, and on October 2, 1 ampule of streptococcus serum together with 10 cc. of a solution of acriflavine in a concentration of 1 to 4,000. With the recognition of the staphylococcus in the culture, the treatment was changed on the morning of October 3, at which tap 5 cc. of staphylococcus bacteriophage (Parke, Davis & Company) and 10 cc. of the acriflavine solution were injected. The injection of bacteriophage was repeated on October 4, 5 and 12. A specimen of spinal fluid taken on October 4 gave a positive culture, but after that all cultures were negative. Three months after the onset of meningitis the boy was attending school, bright and happy but with slight disability in his left leg.

Kulowski [47] reported recovery of a youth aged 18, with lumbosacral osteomyelitis of eight months' duration. After laminectomy meningitis developed, and repeated cisternal punctures yielded purulent fluid containing staphylococci. The patient was discharged with a hopeless prognosis but returned a year later entirely well.

Blitz and Hermann [48] reported the case of a girl of 7 years who had a cold in the head and a stiff neck on February 9 and a temperature of 103.6 F. on February 10. At this time meningococcus serum was injected into the spinal canal. The spinal fluid contained 410 cells per cubic millimeter, and no bacteria were found in the smear. Cultures of the first and third specimens of spinal fluid revealed staphylococci, regarded as possible contaminants by the bacteriologist. Culture of blood taken February 15 yielded staphylococci. On this day 10 mg. of

45. Dmitriev, I. P.: Meningitis Developing After War Wounds, Khirurgiya, 1939, nos. 2 and 3, pp. 111-113.

46. Dunlap, J. E.: Staphylococcic Meningitis with Recovery, J. A. M. A. **104**: 1594-1595 (May 4) 1935.

47. Kulowski, J.: Pyogenic Osteomyelitis of the Spine, J. Bone & Joint Surg. **18**:343-364 (April) 1936.

48. Blitz, R. L., and Hermann, E.: Staphylococcic Septicemia with Meningitis and Multiple Abscesses Treated with Gentian Violet Intravenously: A Case Report, J. Med. **17**:247-248 (July) 1936.

gentian violet (in 5 per cent solution) for each kilogram of body weight was given intravenously, and the same dose was repeated on February 18. The patient was discharged from the hospital February 23 and has remained well. The diagnosis of staphylococcic meningitis seems not to be adequately supported.

Furlow and Reynolds [49] reported success in treating pyogenic meningitis by continuous spinal drainage through a ureteral catheter in situ in the lumbar cistern. Evidently they did not attach great importance to the demonstration of bacteria in the spinal fluid, and on that account their bacteriologic diagnosis would appear not very convincing. In their tabulations they listed 39 cases of staphylococcic meningitis in which treatment was chiefly by multiple lumbar punctures, with 3 recoveries and 36 deaths, and, in addition, 9 cases of staphylococcic meningitis in which lumbar drainage was used, with 6 recoveries.

Wade and O'Neil [50] reported the case of a youth of 18 in whom a low grade pyemia evidently developed from an injury to the right hand. Complicating the pyemia was a purulent spinal meningitis, possibly extradural. Thick pus was obtained by aspiration of the lumbar portion of the spine. After laminectomy of a second and third lumbar vertebrae, incision of the dura with insertion of a drain, and supportive transfusions, the patient recovered in three months.

A recovery from staphylococcic meningitis following treatment with drainage of spinal fluid, transfusions and parenteral administration of fluids, was described by Jackson.[51] The patient was a girl 2½ years old with definite meningitis, smears of the lumbar fluid and cisternal fluid having shown many pus cells and cocci. Cultures of these fluids revealed Staph. aureus, but a culture of the blood did not. The prognosis was considered hopeless. However a transfusion of 250 cc. of blood on July 11 was followed by distinct clinical improvement. After further transfusions there was gradual improvement, with eventual complete recovery.

The patient reported on by Block and Pacella [52] was a boy operated on at the age of 6 days for removal of a meningocele. Ten days later there was a convulsion. Staphylococci were found in the crust at the operative site. The spinal fluid was cloudy. Meningococcus serum was administered, followed by sulfanilamide intrathecally and orally, the total amount

49. Furlow, L. T., and Reynolds, F. C.: Pyogenic Meningitis, South. M. J. **30**:624-632 (June) 1937.

50. Wade, E. M., and O'Neil, F. H.: Staphylococcus Spinal Leptomeningitis, U. S. Nav. M. Bull. **35**:329-331 (July) 1937.

51. Jackson, R. L.: Staphylococcus Aureus Meningitis: Report of a Case with Recovery, J. Pediat. **11**:518-520 (Oct.) 1937.

52. Block, H., and Pacella, B. L.: Staphylococcic Meningitis: Report of a Case in a Seventeen Day Old Infant Successfully Treated with Sulfanilamide, J. A. M. A. **110**:508 (Feb. 12) 1938.

being 10 Gm. The spinal fluid became normal in appearance on the eleventh day of treatment. Unfortunately, the authors did not give the details, particularly in regard to bacteriologic findings.

Fritz and Hollister [53] reported the case of a boy of 13 who was subjected to mastoidectomy on the right side on Jan. 12, 1937 and on the left side on January 16. Staph. aureus was found in the pus from the mastoid. Meningitis developed a few hours after the second operation, and the next morning the spinal fluid contained 24,000 cells per cubic millimeter. Staphylococcus antitoxin was given at once intrathecally and azosulfamide by intramuscular injection. Cultures of spinal fluid taken on the first and second days gave a growth of Staph. aureus, but later specimens were sterile. The injections of antitoxin and of azosulfamide were continued. The patient was discharged from the hospital twenty-seven days after the second operation, totally deaf.

Michels and Gonne [54] reported the case of a boy of 4 years with a discharging ear following measles. On March 28, 1938 meningitis was recognized, and on the next day lumbar tap yielded cloudy fluid. Examination of smears did not reveal any bacteria. A complete mastoidectomy was then done. Culture of the exudate found at operation yielded staphylococci and streptococci. Cultures of spinal fluid showed staphylococci from March 31 to April 29. Administration of sulfanilamide by mouth and by subcutaneous injection, repeated lumbar taps and frequent small transfusions were given credit for the recovery which ensued.

The patient whose case was reported by Pewterbaugh [55] was a boy of 4 years in whom fever and pain in the back developed after an injury on March 3, 1938, which caused an ecchymosis over the first lumbar vertebra. On March 13 the signs of meningitis were definite. Lumbar tap yielded a purulent fluid with many cocci in the smear and Staph. aureus in pure culture. Spinal tap was done every eight hours, and on three occasions cisternal puncture was performed at the same time and the spinal canal was flushed downward with physiologic solution of sodium chloride. Forty cubic centimeters of a 0.8 per cent solution of sulfanilamide was injected into the cisterna magna twice daily. Five grains (0.32 Gm.) of drug was also administered every six hours by mouth. Daily transfusions of blood of 100 cc. each were given for thirteen days beginning on March 14. On March 15 the use of sulfanilamide was discontinued and 40 cc. of staphylococcus antitoxic serum was given

53. Fritz, M., and Hollister, W.: Meningitis Due to Staphylococcus Aureus Haemolyticus: Report of a Recovery, Arch. Otolaryng. **27**:317-322 (March) 1938.

54. Michels, M. W., and Gonne, W. S.: Meningitis Due to Staphylococcus Aureus Haemolyticus Treated with Sulfanilamide, Am. J. Dis. Child. **57**:1379-1385 (June) 1939.

55. Pewterbaugh, D. C.: Staphylococcus Aureus Meningitis, Arch. Pediat. **56**: 623-627 (Oct.) 1939.

intrathecally once a day and 100 cc. was given intravenously twice a day for six days (to March 20). The recovery was credited to the serum.

Another example of staphylococcic meningitis following trauma to the back was reported by Findlay and Hammel.[56] A boy of 14 fell from his bicycle on Nov. 28, 1937 and was admitted to the hospital on December 8 with clinical signs of meningitis. Lumbar tap yielded 30 cc. of purulent fluid. Meningococcus serum was given intrathecally on December 8 and 9. Then the report on culture of the first spinal fluid, indicating the presence of Staph. aureus, caused this serum to be discontinued. Culture of blood taken December 9 was reported also to show Staph. aureus. Sulfanilamide was given to the limit of tolerance, 180 grains (11.66 Gm.) per day by mouth on December 9, 10 and 11. Spinal taps were performed daily from December 8 to December 15, inclusive. The patient was discharged, apparently cured, after twenty-two days.

Fitch [57] has reported the case of a girl of 10 years who suffered a painful injury to her left hip on Nov. 4, 1939. On November 9 she was admitted to the hospital with an abrasion over the left ilium and extreme tenderness over the left lumbar region. After incision of the left lumbar region, stiffness of the neck developed and a positive Kernig sign was noted. Aspiration of the lumbar epidural space yielded thick pus containing staphylococci. Laminectomy was performed. Culture of this pus and culture of the blood showed Staph. aureus. Sulfathiazole was given by mouth from November 14 to November 30 in quantities sufficient to cause cyanosis and cutaneous eruption. Metastatic lesions were incised and drained, and the patient recovered.

Gill [58] reported on a soldier who had sore throat for two weeks and then headache and a convulsion. Lumbar tap yielded a turbid fluid, and the report of its examination in the laboratory indicated 1,070 cells and a prolific growth of staphylococci. Salicylates had been given before the lumbar tap, and after it 2 Gm. of sulfapyridine was administered, followed by 1 Gm. every four hours. When the laboratory report was received the patient was already much improved. All symptoms disappeared in two days.

Dietel and Kaiser [59] reported a case of meningitis in a 20 month old boy which developed a week after a fall resulting in slight injury.

56. Findlay, H. V., and Hammel, M.: Staphylococcus Aureus Meningitis: Report of Case; Treatment with Sulfanilamide—Recovery, California & West. Med. **51**:324-325 (Nov.) 1939.

57. Fitch, T. S. P.: Sulfathiazole in Staphylococcus Aureus Epidural Abscess with Septicemia and Pyemia, Arch. Pediat. **57**:119-124 (Feb.) 1940.

58. Gill, A. M.: Staphylococcal Meningitis Treated with Sulfapyridine: Recovery, Brit. M. J. **1**:810 (May 18) 1940.

59. Dietel, F. W., and Kaiser, A. D.: Staphylococcus Aureus Meningitis Successfully Treated with Sulfathiazole, J. A. M. A. **115**:601 (Aug. 24) 1940.

Lumbar tap on the third day of illness yielded thick creamy fluid containing Staph. aureus. Blood culture revealed the same organism. Administration of sulfathiazole by stomach tube, 2.6 Gm. per day for nine days, and daily spinal punctures with irrigations of physiologic solution of sodium chloride were followed by recovery in three weeks, and the child appeared entirely well two months later.

Lyons,[60] in his report of recoveries from thrombosis of the cavernous sinus, included as his first case that of a woman who had signs of meningitis on admission and positive diagnosis by lumbar tap on the thirty-fourth hospital day after treatment with heparin, sulfapyridine and sulfathiazole since admission. She was discharged with slight ptosis of the eyelid on the seventy-seventh day.

Sadusk and Nielsen [61] reported 1 recovery among the 11 patients with staphylococcic meningitis observed at the New Haven Hospital during twelve years. The patient was a man of 40 who had an astrocytoma removed from his brain on July 10, 1940. Because of staphylococcic infection of the operative wound, a lumbar tap was done on July 17, which revealed normal spinal fluid. On July 19 a second lumbar tap obtained a cloudy fluid which yielded a moderate growth of Staph. aureus in cultures. Sulfathiazole was administered, an initial dose of 4 Gm., followed by 2 Gm. every four hours. The craniotomy wound was opened widely and drains inserted. Sulfathiazole was given intrathecally and intravenously beginning on July 22. Use of the drug was discontinued on July 27 because of toxic fever. The patient was discharged in good condition on August 10 but was still under observation for sequelae at the time of the report.

Cohen and Galpern [62] reported the case of a patient of 29 who was admitted to the hospital on Aug. 6, 1940 in stupor and with a cell count of 5,500 in the spinal fluid. A radical mastoidectomy was performed at once. The patient was then given sulfanilamide, sulfapyridine and sulfathiazole, but these drugs were not well tolerated. Not until the fifteenth postoperative day was there a report of bacteria in the spinal fluid, and this indicated the presence of Staph. albus. After several periods of improvement and relapse the patient was given 100,000 units of staphylococcus antitoxin in divided doses on the forty-second postoperative day, following which there was steady and permanent improvement.

---

60. Lyons, C.: The Treatment of Staphylococcal Cavernous Sinus Thrombophlebitis with Heparin and Chemotherapy, Ann. Surg. **113**:113-117 (Jan.) 1941.

61. Sadusk, J. F., and Nielsen, A. E.: Use of Sulfathiazole in Staphylococcic Meningitis with Recovery, J. A. M. A. **116**:298-300 (Jan. 25) 1941.

62. Cohen, B. M., and Galpern, M.: Sulfathiazole for Treatment of Staphylococcic Meningitis, Arch. Otolaryng. **33**:414-420 (March) 1941.

The case of a 7 month old boy with otogenic staphylococcic meningitis was reported by Donovan.[63] The child was at first given sulfapyridine by mouth and intravenously, and later sulfathiazole was administered. The spinal fluid showed a cell count of 100 on the day of discharge from the hospital. The baby was well three months later.

A recovery from diffuse staphylococcic meningitis in our own series was reported [64] at the Buffalo meeting of the New York State Medical Society, April 29, 1941. The bacteriophage was given by intravenous and by intrathecal injections, and complete recovery resulted.

Street [65] reported a recovery from meningitis in a man aged 28 who had fever and a papular rash on Oct. 27, 1940. Culture of blood taken October 30 revealed growth of Staph. aureus. The neck was stiff, and spinal tap yielded cloudy fluid with a cell count of 150. The cell count in a later specimen was 1,359. There is no mention of culture of the spinal fluid. Sulfathiazole was given by mouth and sulfanilamide by intrathecal and by intravenous injection. The patient recovered without sequelae. The case cannot be accepted because there is no record of a culture of the spinal fluid.

Weary and Lyons [66] reported the case of a youth of 19 in whom staphylococcic meningitis developed after removal of a meningioma. Tension of the wound required aspirations of the right ventricle three or four times a day for four days and less frequently thereafter. On the thirteenth postoperative day lumbar tap yielded a fluid which on culture was found to contain Staph. albus. Sulfathiazole was then started; there was immediate improvement, and the patient was discharged on the thirty-seventh day. These authors reported 3 additional recoveries from staphylococcic meningitis but did not give details.

MacNeal, and Foster [67] reviewed the records of cases of staphylococcic meningitis at the Michigan University Hospital during the fifteen years from 1925 to 1940. There were 17 patients and 17 deaths, although 1 of these patients was discharged from the hospital "cured" after receiving 320,000 units of staphylococcus antitoxin and a thorough course of sulfapyridine therapy, to die at home after a relapse. During the

63. Donovan, T. J.: Staphylococcal Meningitis Treated with Sodium Sulfathiazole, J. Pediat. **18**:518-523 (April) 1941.

64. MacNeal, W. J.: Recent Developments in Bacteriophage Therapy, New York State J. Med. **41**:1531-1536 (Aug. 1) 1941.

65. Street, B.: Staphylococcus Aureus Meningitis with Recovery After Combined Therapy with Sulfanilamide and Its Derivative Sulfathiazole, Minnesota Med. **24**:658-661 (Aug.) 1941.

66. Weary, W. B., and Lyons, J. J. A.: The Chemotherapy of Pyogenic Meningitis with the Sulfonamides, New York State J. Med. **41**:2124-2131 (Nov. 1) 1941.

67. MacNeal, P. S., and Foster, D. B.: Two Cases of Staphylococcic Meningitis Treated with Asparagin Bacteriophage, Am. J. M. Sc. **202**:874-879 (Dec.) 1941.

same period there were 81 patients with "purulent" meningitis without specific bacteriologic diagnosis. One of these patients, a baby of 16 months with a meningocele and "excessive numbers" of leukocytes, recovered promptly after removal of the meningocele. The other 80 died. These authors observed a recovery from staphylococcic meningitis of a man with cerebrospinal syphilis, who evidently became infected with staphylococci by spinal puncture. The patient was treated by intravenous and intrathecal injections of our bacteriophage and therefore will be included in our series of recoveries. Another patient, with both Staph. aureus and Streptococcus viridans in the spinal fluid, was treated with bacteriophage and sulfapyridine. The spinal fluid became sterile, but the patient died.

COMMENT

The significant examples of recoveries from possible staphylococcic meningitis are listed in the table in chronologic order. Doubtless we may have overlooked some valid instances of recovery from staphylococcic meningitis, and some of those listed as not accepted may have been genuine. We also suspect that a few have been admitted to the accepted list which might properly be considered questionable. Roughly, however, we may say that there have been reported in medical literature 48 authentic instances of recovery from staphylococcic meningitis up to Dec. 31, 1941, this list not including the mere announcement of one or several recoveries from this disease for which evidence was not disclosed.

Among the patients who survived there were 21 with the spinal type of staphylococcic meningitis, a form of the disease in which the prognosis would seem to offer more hope. Laminectomy with spinal drainage was employed in treating 14 of the patients who recovered. This surgical procedure merits careful consideration for meningitis primarily involving the spinal region. Here the invasion of the meninges would appear to arise from a vertebral osteomyelitis or other local focus of infection, and in some instances the antecedent inflammation may have contributed to induce some degree of general immunity before the meninges became involved. Among the other patients who recovered, bacterial vaccine was employed for 9, bacterial lysate and bacteriophage for 9 and staphylococcus antiserum for 4. Sulfathiazole was used in 7 cases of recovery, sulfanilamide in 5 and sulfapyridine in 3. In 2 instances sulfonamide compounds appeared unsuccessful and the patients recovered after serum therapy was instituted. In 4 successful cases the patient's skull was opened. Intrathecal lavage was employed to treat 3 patients who recovered.

Staphylococcic meningitis is a malignant disease, but nevertheless it is sometimes not hopeless, either because of slow extension of the infection from a focus adjacent to the meninges or because of low virulence of the particular bacterial strain or relatively increased resist-

Reported Recoveries from Staphylococcic Meningitis

Year	Author	Clinical and Laboratory Notes	Accepted
1893	Macewen [3]	Otogenic; mastoidectomy; no bacteriologic report	No
1897	Luc [3]	Postoperative; further craniotomy; late death	No
1898	Lesné [4]	Infant; spinal drainage; late death	No
1899	Josias and Netter [5]	Meningitis complicating pneumonia; questionable	Yes 1
1899	von Bergmann [6]	Meningocele; operation; meningitis; drainage	Yes 2
1901	Poirier [7]	Cranial trauma; subdural hemorrhage; craniotomy	Yes 3
1901	Barth [8]	Incised wound of spinal dura; laminectomy	Yes 4
1902	Léri [9]	Medical care (Babinski); late death	No
1909	Göbell [10]	Primary spinal type; laminectomy	Yes 5
1909	Kümmel, in discussion on Göbell [10]	Cerebrospinal; multiple taps; data inadequate	No
1912	Preysing [11]	Otogenic; paracentesis and spinal taps	Yes 6
1916	Bourges [12]	Cerebrospinal; spinal taps and fixation abscess	Yes 7
1917	Niles [13]	Staphylococcemia; meningitis (?); data incomplete	No
1920	Rocaz [14]	Meningitis; 1 positive culture in liquid medium	No
1920	Schönbauer and Brunner [15]	Streptococcic (?); staphylococcus vaccine; recovery	No
1920	Lewkowicz [16]	War wound of cranium; staphylococcus vaccine	Yes 8
1920	Lortat-Jacob and Grivot [17]	Otogenic; early mastoidectomy; vaccine	Yes 9
		Otogenic; early mastoidectomy; vaccine	Yes 10
1924	Dandy [19]	Meningitis following lumbar tap; cisternal drainage	Yes 11
1925	Barker [21]	Staphylococcemia; meningitis (?); recovery (?)	No
1925	Sicard and Paraf [22]	Spinal epiduritis; laminectomy	Yes 12
1925	Dupérié, Rocher and Auriat [23]	Lumbar osteomyelitis; laminectomy	Yes 13
1925	Garber [24]	Sacral osteomyelitis; laminectomy	Yes 14
1926	Wharry [25]	Otogenic; inadequate bacteriologic report	No
1926	Moise [26]	Sacral sinus; laminectomy	Yes 15
1927	Emerson [27]	Lumbar osteomyelitis (?); laminectomy	Yes 16
1928	Lamb [28]	Hydrocephalus; intracerebral injection of gentian violet	Yes 17
1928	Baranger [29]	Osteomyelitis; laminectomy; lysate vaccine	Yes 18
1928	Manicatide, Popovici-Lupa and Constantinesco [30]	Intrathecal lavage and vaccine	Yes 19
		Intrathecal lavage and vaccine	Yes 20
1928	Bezançon, Weismann-Netter and Destouches [31]	Lysate vaccine, intrathecally and intravenously	Yes 21
1929	Chavany and George [32]	Spinal type; autogenous vaccine	Yes 22
1929	Benoit [33]	Osteomyelitis; spinal type; vaccine	Yes 23
1930	Sciaroni [35]	Spinal type; laminectomy and drainage	Yes 24
1930	Ballenger [36]	Rhinogenic; craniotomy	Yes 25
1931	Lewis [37]	Cavernous sinus; bacteriophage; late death	No
1931	Stout [38]	Thrombosis of cavernous sinus; intrathecal injection of bacteriophage	Yes 26
1932	Rosamond [39]	Spinal type; laminectomy and drainage	Yes 27
1932	Schless [40]	Diffuse type; intrathecal injection of bacteriophage	Yes 28
1933	Stout [41]	Diffuse type; intrathecal injection of bacteriophage	Yes 29
1934	Bourrel [42]	Skull fracture; intrathecal injection of serum; electrargol	Yes 30
1935	Arauz and Tato [43]	Otogenic; mastoidectomy; cinchophen-methenamine preparation	No
1935	Barthélemy [44]	Gunshot wound of brain; bactéstaphyphage	Yes 31
1935	Dunlap [46]	Apparently diffuse; intrathecal injection of bacteriophage	Yes 32
1936	Kulowski [47]	Lumbar osteomyelitis; laminectomy	Yes 33
1936	Blitz and Hermann [48]	Staphylococcemia; intravenous injection of gentian violet	No
1937	Furlow and Reynolds [49]	Pyogenic meningitis; spinal drainage	No
1937	Wade and O'Neil [50]	Spinal type; laminectomy	Yes 34
1937	Jackson [51]	Infant; transfusions and spinal taps	Yes 35
1938	Block and Pacella [52]	Infant; infected meningocele; sulfanilamide	No
1938	Fritz and Hollister [53]	Mastoidectomy; antitoxin; azosulfamide	Yes 36
1939	Michels and Gonne [54]	Mastoidectomy; sulfanilamide; transfusions	Yes 37
1939	Pewterbaugh [55]	Injury to back; spinal irrigation; antitoxin	Yes 38
1939	Findlay and Hammel [56]	Diffuse; following trauma; sulfanilamide	Yes 39
1940	Fitch [57]	Spinal type; laminectomy; sulfathiazole	Yes 40
1940	Gill [58]	Sulfapyridine; cure in two days	No
1940	Dietel and Kaiser [59]	Spinal type; thecal irrigations; sulfathiazole	Yes 41
1941	Lyons [60]	Thrombosis of cavernous sinus; heparin; sulfonamide compounds	Yes 42
1941	Sadusk and Nielsen [61]	Craniotomy; intrathecal injection of sulfathiazole	Yes 43
1941	Cohen and Galpern [62]	Mastoidectomy; treatment with sulfonamide compounds a failure; antitoxin	Yes 44
1941	Donovan [63]	Otogenic; sulfapyridine and sulfathiazole	Yes 45
1941	MacNeal [64]	Diffuse meningitis; bacteriophage	Yes 46
1941	Street [65]	Meningitis; no culture; sulfonamide compounds	No
1941	Weary and Lyons [66]	Craniotomy; 1 positive culture; sulfathiazole	Yes 47
1941	MacNeal and Foster [67]	Diffuse type; intrathecal and intravenous injections of bacteriophage	Yes 48

ance of the host. It also appears that very young children offer a better prognosis. Perhaps the most dramatic example in the series was afforded by the 16 year old girl whose case was the first reported by Stout [38] (1931). This patient, with thrombosis of the cavernous sinus, staphylococcemia and abundant staphylococci in the turbid spinal fluid, could not have been accorded an optimistic prognosis by any stretch of the imagination.

SUMMARY

Forty-eight recorded recoveries from staphylococcic meningitis have been found in the medical literature published from 1893 through 1941.

The relatively localized spinal form of the disease offers the best prognosis, especially in young persons. Surgical drainage by laminectomy has been successful in 14 of these cases.

Bacterial vaccines have been credited with some recoveries in the more chronic types of the disease.

There are reports of successful treatment of the acutely invasive forms of staphylococcic meningitis by use of sulfonamide compounds and by use of bacteriophages.

TREATMENT OF VICTIMS OF POISON GAS

WITH SPECIAL REFERENCE TO THE USE OF THE BRONCHOSCOPE

HORACE E. MITCHELL, M.D.
LAKEWOOD, OHIO

The treatment of victims of poison gas is a subject that members of the medical profession, particularly otolaryngologists, should be thinking about and investigating at this time. The available literature offers considerable food for thought and points the way to some of the preparations and plans that we in this country should be making if the need to cope with this emergency should arise. However remote gas warfare now seems, it is a possibility we must face, because the Japanese have used poison gas, particularly lewisite, on the Chinese, and they will do the same to us, if they ever get the chance; the Germans, moreover, may be forced to resort to gas warfare as a measure of desperation.

It is not the purpose of this paper to present in detail the various phases of the problem with which one should familiarize oneself. These include the different types of chemicals used as poison gases, the characteristic clinical symptoms produced by each type, the clinical classification of severe (asphyxiant) gas poisoning, preventive and disciplinary measures, first aid methods and, finally, the medical treatment of victims of gas poisoning. A comprehensive discussion of these factors is available in the "Technical Manual" issued by the United States War Department.[1] It seems apropos, rather, to treat these factors in a general way, to show how the treatment of gas poisoning is of primary concern to the otolaryngologist and to stress the importance of the use of the bronchoscope, advocated by the Jacksons,[2] which has been completely ignored in general articles on the subject.

CLASSIFICATION OF GASES

The principal poison gases have been classified by Underhill[3] into two groups: neutralizing gases, so called because their purpose is not to kill, but to neutralize the effectiveness of military resistance, and the

1. Treatment of Casualties from Chemical Agents, Technical Manual 8-285, United States War Department, 1941.

2. Jackson, C., and Jackson, C. L.: Bronchoscopy, Esophagoscopy and Gastroscopy, ed. 3, Philadelphia, W. B. Saunders Company, 1934.

3. Underhill, F. P.: The Lethal War Gases: Physiology and Experimental Treatment, New Haven, Conn., Yale University Press, 1920.

lethal gases, or asphyxiants, which may destroy life. These two groups have also been referred to as harassing gases and lung irritants. The principal lethal, or asphyxiant, gases are chlorine, phosgene (carbonyl chloride) and chloropicrin. Nitric acid vapors and ammonia also are listed by the War Department. In addition, irritation of the lungs may result from some of the so-called nonlethal gases, and their most serious sequelae are due to this effect.

The harassing gases include vesicants, or blistering agents, such as mustard gas (dichloroethylsulfide), lewisite (dichlorobetachlorovinylarsine) and ethyldichloroarsine; lacrimators, or tear gases, such as chloroacetophenone and benzyl bromide, and irritant smokes, or sternutators, such as adamsite (diphenylaminearsine chloride) and diphenylchloroarsine. In addition, screening smokes, such as white phosphorus, incendiary agents, systemic poisons and incidental gases, such as carbon monoxide, nitric acid fumes and ammonia, resulting from bombing, may produce casualties in warfare.

FIRST AID MANAGEMENT

Although it is impossible to discuss first aid management of casualties resulting from all these agents,[4] it may be well to point out the chief measures to be taken in case of mustard gas and phosgene poisoning, since these two gases are the most likely to be encountered. Mustard gas is a blistering agent, or vesicant, which produces lesions on the skin as well as the mucous membranes, and phosgene is the principal asphyxiant, or lethal gas.

The main features of the first aid management of victims of mustard gas, as compiled by the *British Medical Journal*[5] from government regulations, include: (1) removal of outer garments and shoes; (2) methods directed toward the saving of life and treatment of shock; (3) saving of the eyes, and (4) cleansing of the skin.

The requirements of first aid treatment of victims of phosgene poisoning are as follows: 1. Every person, regardless of symptoms, who has been exposed to an asphyxiant gas should be placed on a stretcher as soon as possible. 2. Cases should be classified immediately at the hospital or the treatment center as instances of mild, "blue" or "gray" poisoning. 3. Oxygen should be administered to patients with severe poisoning. 4. Even patients with the slightest symptoms should be under observation and at rest for at least forty-eight hours. The most important point to bear in mind is that any person, regardless of symptoms or the

4. Serra, G.: First Aid for Victims of Gas, Gior. di. med. mil. **88**:530 (July) 1940; abstracted, War Med. **1**:114 (Jan.) 1941.

5. Diagnosis and Treatment of Gas Casualties, Brit. M. J. **1**:723-724 (May 10) 1941.

lack of them, who has been exposed to phosgene without a gas mask should not be allowed to move about, but should be placed on a stretcher at the earliest possible moment.

SYMPTOMS

The symptoms caused by mustard gas are more or less typical for the whole group of nonlethal gases. Inflammation and lesions of the upper respiratory tract, with possible bronchitis, laryngitis and pneumonia, are the predominant symptoms of most gas poisoning, as can be seen from the tables in the Manual of the War Department. The early symptoms of mustard gas poisoning are cutaneous burns, particularly of the softer and moist surfaces; blepharitis and conjunctivitis; laryngitis, tracheitis and bronchitis, and vomiting. The sequelae, in cases of the graver type, include corneal ulceration, bronchopneumonia and bronchiectasis.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of gas poisoning is presented in detail in the "Technical Manual" of the War Department. This involves a careful history, including data especially on the odor of the gas inhaled, the length of the exposure and the length of time and the degree of activity since the exposure. The physical examination should note the patient's appearance, whether or not there is cyanosis, any engorgement of the veins of the neck, the type and quantity of sputum, the presence or absence of cough and pain in the chest and the results of auscultation of the chest. The prognosis is good, as a rule, unless there is pulmonary edema, and all postmortem examinations in fatal cases have shown the presence of edema of the lungs.

CLINICAL CLASSIFICATION OF ASPHYXIANT GAS POISONING

The clinical classification of asphyxiant forms of gas poisoning is of extreme importance in organization of effective countermeasures, for the patients must be cared for in the order of urgency, that is, according to the degree of respiratory difficulty and cyanosis. The condition is classified as mild when there is slight dyspnea; moderate, or "blue," when there are pronounced respiratory distress and edema, and severe, or "gray," when there are pronounced edema, inability to breathe and a shocklike state. The "gray" form is the most serious and hence must receive the most immediate attention. All patients who have been exposed to phosgene or to an asphyxiant gas mixture should be watched carefully, for they sometimes proceed from one stage to the next within a short time.

There is some disparity of opinion regarding the relative importance of the administration of oxygen in cases of severe asphyxiant gas poison-

ing. Underhill, whose conclusions were based on clinical studies begun in the last war, followed by exhaustive animal experimentation, expressed the belief that the cause of death is the concentration of the blood and not the edema per se, and hence that the introduction of fluid is of prime importance. He expressed the belief that the administration of oxygen is a helpful adjuvant, but did not think that it would save life, without reduction of the concentration of the blood. On the other hand, the British medical authorities have cautioned against venesection with infusion of fluids or transfusion, on the basis that the procedure aggravates the edema of the lungs. The United States War Department recommends bleeding and the administration of oxygen.

USE OF THE BRONCHOSCOPE IN CASES OF ASPHYXIANT GAS POISONING

Some statements by the Jacksons [2] are extremely pertinent to this controversy and may provide the clue to the solution of the problem. They pointed out that "inhalations of flames and fumes produce quantities of a serous exudate from which the victims drown in their own secretions; a condition often mistaken for edema of the lungs or pneumonia." That this interpretation is correct is indicated by the fact cited by other authors, that the "edema" is so severe that the patient may be emitting foam from the mouth. Jackson and Jackson stated that such a patient requires bronchoscopic aspiration of secretions, before insufflation of oxygen, which can be introduced through the bronchoscope.

Those who have been writing about the treatment of war gas poisoning have not referred to the possible use of the bronchoscope in these cases of severe respiratory involvement, probably because they have approached the problem from some other standpoint and are not familiar with bronchoscopic technic. As otolaryngologists, the possibility of this approach should engage our attention, because, as the Jacksons have pointed out, it has certain obvious advantages over other methods of artificial respiration. In the first place, there is no contraindication to its use, as with other methods, because of fracture of the ribs, clavicle or spine. The oxygen mixture (with 10 per cent carbon dioxide) necessary is available in every hospital. Bronchoscopy can be carried out in a few minutes by a skilful technician, and it is especially easy in cases of respiratory arrest, because of the limp condition of the patient.

Jackson and Jackson described the method as follows:

> The bronchoscope is passed through the laryngoscope and aspiration of the secretion is accomplished by the use of the long aspirating tube through the bronchoscope. The oxygen is turned on at the tank and the flow regulated before the rubber tube from the wash-bottle is attached to the side-outlet of the bronchoscope (usually used for aspiration). It is necessary to be certain that the flow is gentle, so that, with a free return flow the introduced pressure does not exceed

the capillary pressure; otherwise the blood will be forced out of the capillaries and the ischaemia of the lungs will be fatal. Another danger is that overdistention causes inhibition of inspiration resulting in apnea continuing as long as the distention is maintained, if no longer. The return flow from the bronchoscope should be interrupted (by compressing the rubber tube with the thumb and finger) for two or three seconds several times a minute to inflate the lungs, but the flow must not be occluded longer than three seconds because the intrapulmonary pressure would rise. A pearl of amyl nitrite may be broken in the wash bottle. Slow rhythmic artificial respiratory movements are a useful adjunct, and unless the operator is very skillful in gauging the alternate pressures and releases with the thumb according to the oxygen pressure, it is vitally necessary to fill and deflate the lungs rhythmically by one of the well known methods of artificial respiration.

They went on to say:

The pulmotor and similar mechanisms are, perhaps, the best things the use of which can be taught to laymen; but as compared to bronchoscopic oxygen insufflation they are woefully inefficient, because the intraoral pressure forces the tongue back over the laryngeal orifice, obstructing the airway in this "death zone." By the introduction of the bronchoscope this death zone is entirely eliminated, and a free airway established for piping the oxygen directly into the lungs.

In addition to the advantages stressed by the Jacksons, the bronchoscope allows the operator to see the larynx and the tracheobronchial tree, so that he can get direct evidence of laryngeal edema that might be causing obstructive dyspnea. In cases of such a condition the necessary tracheotomy could be performed over the bronchoscope.

Additional comments on the bronchoscopic treatment of asphyxiant gas poisoning are contained in a letter recently received from Dr. Jackson:

I am glad to note that you are spreading the gospel of bronchoscopic aspiration and insufflation. These are life-saving measures of utmost importance in all forms of impending asphyxia. All the statements made . . . were based on clinical experience in Pittsburgh and Philadelphia, largely with industrial casualties. This demonstrated that in all cases of inhalation of smoke, fumes and irritating or asphyxiating gases the best treatment is bronchoscopic insufflation of oxygen, with aspiration of secretions. It is additionally helpful, as well as comforting, to spray in a weak solution of sodium bicarbonate. This has the effect of lessening the viscosity of secretions, thus facilitating their removal not only by bronchoscopic aspiration but afterward by the physiologic method of cough.

In World War I the bronchoscope was not used except in a few general hospitals, and there only in cases of foreign bodies. No equipment was available nearer the front in any country. In the present war equipment has so far been inadequate, and reports are scant as yet. The indications are that ample bronchoscopic equipment will be available as near the front as feasible, as well as for civilian casualties.

If the "edema of the lungs" in cases of severe asphyxiant gas poisoning is, for the most part, due to fluid exudate, which can be aspirated,

then the objection of certain authorities to the use of infusions or transfusions because of the fear of aggravating the edema is no longer valid, and this measure, designed to reduce the concentration of the blood, can be applied along with administration of oxygen to the lungs. It seems also that it would be worth while to experiment with the use of heparin, which might help to reduce the viscosity of the blood.

GENERAL TREATMENT

After the appropriate emergency measures and prophylactic treatment have been directed toward prevention of the more serious later sequelae, rest and general nursing care, with suitable palliative and local treatment of the areas affected, particularly of the mucous membranes, are carried out, as for comparable lesions produced by other agents.[6]

COMMENT

In case of mass casualties from asphyxiant gas poisoning, no doubt it would be impossible to use the bronchoscope in many instances. But the instrument certainly should have a prominent place in the plans and preparations for meeting such an emergency, and it is the duty of otolaryngologists to acquaint other members of the medical profession with its life-saving potentialities.

As otolaryngologists we have a grave responsibility in the management of gas casualties because the most prominent symptoms of all types of gas poisoning are related to the upper and the lower respiratory tract. Since we are bound to be concerned, should the need arise, with the ultimate treatment of the victims of gas poisoning, it is our duty to learn as much as possible about all phases of the problem and to assume initiative and leadership in organizing the necessary facilities to cope with it.

14805 Detroit Avenue.

6. Eckert-Möbius, A.: Diseases of Upper Respiratory Tract Due to Poison Gases and Their Treatment, Med. Welt **14**:313 (March 30) 1940; abstracted, War Med. **1**:115-116 (Jan.) 1941. Footnote 1.

OSTEITIS FIBROSA CYSTICA: DIFFERENTIAL DIAGNOSIS

WITH NOTE ON REPAIR OF A MAXILLARY LESION BY CARTILAGINOUS ISOGRAFT

NOAH FOX, M.D.
AND
VITO TAGLIA, M.D.
CHICAGO

Skeletal deformities about the head are frequently the first manifestation of generalized osseous disease; hence the otolaryngologist is often called on to diagnose these syndromes.

In 1891 von Recklinghausen [1] described in detail the condition which bears his name. He believed the disease resembled that described by Engel [2] in 1864 and Langendorff [3] in 1877. In 1884 Davies-Colley [4] described a typical case of osteitis fibrosa cystica. Erdheim [5] was the first to suggest the relationship of the parathyroid glands to this condition. He believed, however, that the changes in the parathyroid were secondary and that changes in the bone were primary. Mandl [6] in 1925 transplanted parathyroid tissue into the abdominal wall of a man who had this disease, hoping to prove Erdheim's theory. Nothing resulted from this procedure, but an exploratory operation on the neck revealed a parathyroid tumor, proving the causation of this disease.

From the Department of Otology, Rhinology and Laryngology of the University of Illinois College of Medicine.

1. von Recklinghausen, F.: Die Fibrose oder deformirende Ostitis, in Festschrift Rudolf Virchow zu seinem 71. Geburtstage gewidmet von den früheren und jetzigen Assistenten des Berliner, pathologischen Instituts, Berlin, G. Reimer, 1891, pp. 1-39.

2. Engel, G.: Ein Fall von cystoider Entartung des gesammten Skelets, Thesis, Giessen, F. C. Pietsch, 1864.

3. Langendorff, O., and Mommsen, J.: Beitrag zur Kenntnis der Osteomalazie, Virchows Arch. f. path. Anat. **69**:442-487, 1877.

4. Davies-Colley, N.: Juvenile Osteomalacia, Brit. M. J. **1**:667, 1884.

5. Erdheim, J.: Ueber Epithelkörperbefunde bei Osteomalacie, Sitzungsb. d. k. Akad. d. Wissensch. Math-naturw. Cl. **116**:311-370, 1907.

6. Mandl, F.: Therapeutische Versuch bei einem Falle von Ostitis fibrosa generalista mittels Exstirpation eines Epithelkörperchentumors, Zentralbl. f. Chir. **53**:260-264 (Jan.) 1926.

Though this condition has been considered comparatively rare, Wilder and Howell [7] by 1936 were able to collect 135 cases. In the past five years numerous other cases have been reported.

Various classifications of hyperparathyroidism occur in the literature. Albright and his associates [8] have suggested differentiating this disease as follows: (1) primary hyperparathyroidism due to an adenoma; (2) primary hyperparathyroidism due to an idiopathic hyperplasia; (3) renal disease causing compensatory hyperplasia. Helfet [9] suggested the following classification:

1. Primary hyperparathyroidism—due to an adenoma and typically represented by generalized fibrocystic disease, with a raised blood calcium content and reduced blood phosphorus.

2. Secondary hyperparathyroidism.—*a.* Simple, compensatory or physiological, resembling a mild picture of (1) but with a normal blood calcium and blood phosphorus. Parathyroid tissue is normal or shows general hyperplastic changes only. *b.* Pathological—where adenoma has been superimposed on (2 *a*). Clinically similar to (1) but reverting to (2 *a*) after removal of the adenoma.

ETIOLOGY

Osteitis fibrosa cystica is a disease of middle life and affects women about twice as often as men. It is bilateral and is always associated with parathyroid tumor, the oversecretion of which causes the symptom complex. According to Kyser [10] the following applicable physiologic data have been accumulated:

1. The sole function of vitamine D is to aid in the absorption of calcium from the intestine. 2. The parathyroid hormone raises the concentration of serum calcium and lowers the concentration of inorganic phosphorus of the serum. 3. It is thought that the parathyroid hormone causes increased activity of the phosphatase of the serum. The increased activity of this enzyme causes direct removal of calcium from bone; thus the level of serum calcium is raised.

PATHOLOGY

Associated as it is with hyperactivity of parathyroid adenomas, there results a decided disturbance of the calcium and phosphorus metabolism. Great quantities of calcium are released from the bones, which results

7. Wilder, R. M., and Howell, L. P.: Etiology and Diagnosis in Hyperparathyroidism: Review of One Hundred and Thirty-Five Proved Cases, J. A. M. A. **106**:427-431 (Feb. 8) 1936.

8. Albright, F.; Drake, T. G., and Sulkowitch, H. W.: Renal Osteitis Fibrosa Cystica, Bull. Johns Hopkins Hosp. **60**:377-399 (June) 1937.

9. Helfet, A. J.: A New Conception of Parathyroid Function and Its Clinical Application, Brit. J. Surg. **27**:651-677 (April) 1940.

10. Kyser, F. A.: Hyperparathyroidism, Proc. Staff Meet., Mayo Clin. **15**: 179-181 (March 20) 1940.

in a high serum calcium level. Similarly, larger amounts of calcium are found in the feces and urine than can be accounted for by ingestion of calcium. This negative calcium balance, as well as high serum calcium, is characteristic of the disease. Because of an increase in the phosphatase activity of the serum, there is a decrease in the serum phosphorus as well as the excreted phosphorus of the urine and feces.

According to Albright and his associates,[11] absence of hypophosphatemia may be explained in some cases by the presence of renal impairment. Also, a normal amount of serum calcium may be present during a remission. In 28 cases in the literature cited by Gutman and his associates,[12] the phosphatase activity was found to be increased in every instance. However, the increase in phosphatase, according to Bodansky and Jaffe [13] and Franseen and his associates,[14] is dependent on osteoblastic activity and is directly proportional to the extent and intensity of the attempt at ossification, which may not be particularly prominent in osteitis fibrosa cystica. The levels of calcium and phosphorus in the blood, as well as the values obtained in determinations of phosphorus and calcium balance, are of greater value in diagnosing osteitis fibrosa cystica than is the value for blood phosphatase. Other laboratory findings include a high nonprotein nitrogen content and hypochromic anemia.

The changes in the tissues are referable to the parathyroid, which presents the changes of a benign adenoma, and to the skeletal system, which presents the picture of cystic degeneration and fibrosis. The skeletal changes, which involve all bones bilaterally, include expansion, softening and thinning of the cortical bone. The medullary cavity is filled with a pinkish, granular fibrous tissue. Cysts may be present, and the bone cuts easily. Except where callus formation occurs at the site of a fracture, no new bone is seen.

Histologically, lacunar resorption occurs with fibrous replacement of the normal osseous architecture. The fibrous tissue is rather cellular,

11. Albright, F.; Aub, J. C., and Bauer, W.: Hyperparathyroidism: Common and Polymorphic Condition as Illustrated by Seventeen Proved Cases from One Clinic, J. A. M. A. **102**:1276 (April 21) 1934.

12. Gutman, A. B.; Tyson, T. L., and Gutman, E. B.: Serum Calcium, Inorganic Phosphorus, and Phosphatase Activity in Hyperparathyroidism, Paget's Disease, Multiple Myeloma and Neoplastic Disease of the Bones, Arch. Int. Med. **57**:379-413 (Feb.) 1936.

13. Bodansky, A., and Jaffe, H. L.: Phosphatase Studies: III. Serum Phosphatase in Diseases of the Bone; Interpretation and Significance, Arch. Int. Med. **54**:88-110 (July) 1934.

14. Franseen, C. C.; Simmons, C. C., and McLean, R.: The Phosphatase Determination in the Differential Diagnosis of Bone Lesions, Surg., Gynec. & Obst. **68**:1038-1046 (June) 1939.

with both young and matured fibroblasts present. It is relatively avascular and contains a large amount of collagen. There are many spindle cells containing pale-staining nuclei. There is some lymphocytic infiltration with occasional nests of large osteoclasts present. Osteoblastic activity is almost absent, though an occasional small deposit of poorly formed bone or cartilage is noted. In some places, the matrix has the appearance of lacunas, but the trabeculae appear faint, and little calcium is present.

SYMPTOMATOLOGY

The symptoms, like the pathologic features, are due for the most part to changes referable to hyperactivity of the parathyroids. Kyser [10] quoted Shelling's [15] description of the symptoms as follows:

1. Symptoms due to hypercalcemia.
   - (1) Muscular weakness.
   - (2) Lassitude.
   - (3) Hypotonia.
   - (4) Chronic constipation.
   - (5) Slow pulse, and cardiac arrythmias.
2. Symptoms related to the skeletal system.
   - (1) Pain referable to the bones and joints of the extremities and spine.
   - (2) Cystic regions in the long bones and skull.
   - (3) Spontaneous fracture.
   - (4) Kyphosis, scoliosis and deformity of the thorax.
   - (5) Deformities of the long bones.
   - (6) Waddling gait or inability to walk.
3. Symptoms due to transportation and excretion of calcium.
   - (1) Polyuria and polydipsia.
   - (2) Enuresis, nocturia and dysuria.
   - (3) Renal complications (stones).

ROENTGENOLOGIC FINDINGS

Roentgenologically, the characteristic changes are those of a coarsely granular osteoporosis due to the loss of calcium with replacement by fibrous or cystic tissue. In addition are seen deformities of the bones due to collapse of supporting structures. The long bones present a broadening or expansion with a thinning of the cortex where cysts are present. The texture of the bone appears rarefied and trabeculated. Between the cystic areas, the bone shadows appear diminished in density.

15. Shelling, D. H.: The Parathyroids in Health and in Disease, St. Louis, C. V. Mosby Company, 1935; cited by Kyser.[10]

DIFFERENTIAL DIAGNOSIS

The condition most difficult to differentiate from osteitis fibrosa cystica is polyostotic fibrous dysplasia. Of this condition Lichtenstein,[16] who described it, stated:

> The name "polyostotic fibrous dysplasia" is being used in the Hospital for Joint Diseases to designate a skeletal developmental anomaly affecting several or many bones with predominantly unilateral involvement. The involved bones show filling of their medullary cavities by gritty, grayish white fibrous tissue containing trabeculae of newly formed primitive bone. The condition apparently results from perverted activity of the specific bone-forming mesenchyme. It manifests itself early in adult life or in childhood and evolves slowly, pursuing a protracted clinical course characterized by pain, deformity and a tendency to pathologic fracture of the affected bones.

This condition, though so closely resembling osteitis fibrosa cystica in its symptoms and pathologic picture, differs in that polyostotic fibrous dysplasia is associated with normal values for serum phosphorus and calcium as well as a normal calcium and phosphorus balance. Values for phosphatase activity are high, and there is, of course, no associated parathyroid tumor.

H. L. Jaffe[17] said:

> One important point in the roentgenologic differential diagnosis between "polyostotic fibrous dysplasia" and hyperparathyroidism is rarefaction of the cortex in most bones in the latter and the absence of cortical rarefaction except in the affected bone in the former.

The condition described by Adams and his associates[18] as "regional fibrocystic disease" seems to vary in no great wise from that described by Lichtenstein as polyostotic fibrous dysplasia.

Osteitis fibrosa disseminata, or osteodystrophia fibrosa, is another condition closely resembling von Recklinghausen's disease. Albright and his associates[19] suggested the former term to differentiate the condition from osteitis fibrosa generalisata (hyperparathyroidism) and osteitis fibrosa localisata (solitary bone cyst). They summarized the findings of this condition as follows:

> (*a*) Multiple bone cysts which have a distribution suggesting a relation to nerve roots or to an embryologic defect in the myotomes.

---

16. Lichtenstein, L.: Polyostotic Fibrous Dysplasia, Arch. Surg. **36**:874-898 (May) 1938.

17. Jaffe, H. L., in discussion on Garlock, J. H.: The Differential Diagnosis of Hyperparathyroidism with Special Reference to Polyostotic Fibrous Dysplasia (Lichtenstein-Jaffe), Ann. Surg. **108**:347-361 (Sept.) 1938.

18. Adams, C. O.; Compere, E. L., and Jerome, J.: Regional Fibrocystic Diseases, Surg., Gynec. & Obst. **71**:22-32 (July) 1940.

19. Albright, F.; Allan, M. B.; Hampton, A. O., and Smith, P.: Syndrome Characterized by Osteitis Fibrosa Disseminata, Areas of Pigmentation and Endocrine Dysfunction, with Precocious Puberty in Females, New England J. Med. **216**:727-745 (April 29) 1937.

(*b*) Areas of pigmentation which have a distribution suggesting some connection with the bone cysts.

(*c*) Precocious puberty in females but apparently not in males.

It should be added that this condition is only slowly progressive and usually starts before puberty. The blood chemistry is normal except for abnormal values for cholesterol, fat and sugar. Serum phosphatase is usually elevated in advanced stages.

Other tumors causing hypercalcemia must be differentiated. These include multiple myeloma and metastatic carcinoma.

Multiple myeloma is associated with the presence of Bence Jones proteins in the urine in 80 per cent of the cases.[20] Roentgenologically, the bone appears to be moth-eaten. Histologically, the picture is that of a proliferation of some one cell of the marrow.

Bone metastases from primary tumors may be osteoplastic or osteolytic.[21] The latter type causes a marked lacunar absorption and destruction of bone, so that on roentgen examination an extreme porosis is noted. It resembles the lesion of von Recklinghausen's disease, particularly where expansion of the bone by the tumor mass has occurred. Histologically, however, the characteristics of the mother tumor are revealed.

Osteitis deformans, or Paget's disease, must be differentiated from this condition. Paget's is a disease of middle life. It is characterized by an invasion of the bone by connective tissue. The vascular elements of the haversian canals and bone marrow become converted into connective tissue around which new bone is deposited. This results in a narrowing of the marrow cavity and a thickening of the bony cortex, causing an increase in the size of the bones.[22]

Roentgenologically, the involved bones appear coarsely mottled with accentuation of the trabeculae and enlargement of the cortical portion. The picture is that of a marked hyperostosis and sclerosis. The very high osteoblastic activity present in this disease is reflected in a high serum phosphatase content, the only consistent abnormality of the blood.

Osteomalacia, or adult rickets, though rare in this country, also resembles osteitis fibrosa cystica. This condition occurs most frequently in very poorly nourished women during the child-bearing period. It is definitely a deficiency disease due to a lack of the fat-soluble vitamin D, resulting in a demineralization of the bones.[23] Vitamin D fixes calcium

20. Charlton, T. J.: Multiple Myeloma, Arch. Int. Med. **40**:98-109 (July) 1927.

21. Moore, A. B.: Roentgenologic Study of Metastatic Malignancy of Bones, Am. J. Roentgenol. **6**:589-593 (Dec.) 1919.

22. Nicholas, D. R.: Osteitis Deformans (Paget's Disease of Bone): Report of Case, Proc. Staff Meet., Mayo Clin. **15**:182-184 (March 20) 1940.

23. Maxwell, J. P.: Further Studies in Adult Rickets, Proc. Roy. Soc. Med. **32**:287-297 (Feb.) 1939.

and phosphorus, thus decreasing endogenous elimination. It also promotes intestinal absorption of these minerals, making available large quantities of calcium and phosphorus for the bones.

Roentgenologically, the bones in osteomalacia reveal a severe osteoporosis, with fewer striations than are seen in osteitis fibrosa cystica but with involvement about the epiphyseal ends of the long bones.

The pathologic changes are similar to those noted in childhood rickets, i. e., a softening of the bone, with the formation of cystic cavities. Microscopically, there is a loss of bone tissue, with newly formed bone having wide haversian canals with little calcium and a marked tendency to formation of osteoid tissue.

Chemical examination of the blood reveals a low serum calcium and phosphorus with a slight increase in phosphatase.

Osteogenesis imperfecta tarda, or marble bone disease, must also be differentiated. Other synonyms for this disease are: osteopsathyrosis congenita, fragilitas ossium congenita, Lobstein's disease, idiopathic osteopsathyrosis, Albers-Schönberg disease, mollities ossium and periosteal dysplasia. Characteristic symptoms are brittle bones, blue sclerotics, deafness and relaxation of ligaments.

The classification of idiopathic fragility presented by Hills and McLanahan,[24] which is a modification of Babcock's,[25] is as follows:

I. Hereditary type. Hereditary hypoplasia of the mesenchyme (brittle bones and blue scleras)

II. Nonhereditary congenital type
  1. Osteogenesis imperfecta congenita
  2. Osteogenesis imperfecta tarda (osteopsathyrosis)
    (1) With white scleras
    (2) With blue scleras

III. Nonhereditary acquired type.
  1. Osteosclerosis fragilis generalisata (marble bones or Albers-Schönberg disease)

IV. Senile type
  1. Osteoporosis

Roentgenographic study reveals marked decalcification of all bones, with numerous fractures and bowing of long bones. There is a thinning of the cortex with narrowing of the shaft. Nodular spotted areas of increased density appear in the spongiosa, and linear striae or streaks of condensation appear along the shaft.

24. Hills, R. J., and McLanahan, S.: Brittle Bones and Blue Scleras in Five Generations, Arch. Int. Med. **59**:41-55 (Jan.) 1937.

25. Babcock, W. W.: A Textbook of Surgery for Students and Physicians, Philadelphia, W. B. Saunders Company, 1935, p. 203.

Pathologic examination reveals a deficiency in ossification of the calcified cartilage columns, resulting in distortion of the trabecular pattern. The periosteum of the diaphyses is thickened, but the cortex is thin, discontinuous or lacking. The trabeculae are incompletely calcified, are diminished in number and stain unevenly. The medulla is fibrous or lymphoid.

The calcium and the phosphorus content of the blood are normal, though phosphatase may be increased.

Renal rickets commonly causes disturbances in the long bones, osteoporosis and granular mottling, particularly in the skull. In this condition there is a long history of renal impairment which preceded the bony changes.

Roentgenologic studies reveal a mottling or woolly appearance of the bones. Characteristically, there is noted a calcification of the medium-sized and small arteries.

The pathologic change, according to Shelling and Remsen,[26] consists of a fibrous displacement of the bone tissue. The tissue contains numerous giant cells, blood vessels, poorly calcified spicules of bone without osteoid borders and also many cystlike areas of microscopic size.

The kidneys show extreme changes, with long-standing hydronephrosis. Frequently little normal kidney tissue remains. A simple hyperplasia of the parathyroid occurs. Metastatic calcification of the media of systemic arteries, the Mönckeberg type of arteriosclerosis, also occurs.

Chemical determinations reveal a marked renal insufficiency with a particularly high nonprotein nitrogen content. There is a decided retention of phosphorus, with a reduction in serum calcium. Elimination of both phosphorus and calcium occurs through the bowel because of impairment of kidney function.

Ollier's disease, or dyschondroplasia, may also resemble osteitis fibrosa cystica. This condition was first described by Ollier [27] in 1899 as "cartilaginous dystrophy." It is characterized by the presence of areas of cartilage in the shafts of the long bones, especially near the growing ends. It is called Ollier's disease when it involves only one

26. Shelling, D. H., and Remsen, D.: Renal Rickets: Report of a Case Showing Four Enlarged Parathyroids and Evidence of Parathyroid Hypersecretion, Bull. Johns Hopkins Hosp. **57**:158-181 (Sept.) 1935.

27. Ollier: De la dyschondroplasie, Bull. Soc. de chir. de Lyon **3**:22, 1899; cited by Sanderson, G. H., and Smyth, F. S.: Chondrodysplasia (Ollier's Disease): Report of a Case Resembling Osteitis Fibrosa Cystica, J. Bone & Joint Surg. **20**:61-67 (Jan.) 1938.

side of the body and dyschondroplasia when it involves both sides of the body symmetrically.

Roentgenologically, these areas appear cystic, as in osteitis fibrosa cystica. The ends of the diaphyses of the long bones present longitudinal striations and gouged-out areas. Where new calcium is laid down these gouged-out areas appear spotted. Bentzon [28] has described fan-shaped striations in the wings of the iliums. There is never any involvement of the carpal or tarsal bones. The juxtaepiphysial distribution, broad metaphyses and exostoses are characteristic of Ollier's disease.[29]

The blood chemistry is normal.

Other less common conditions which might be confused include pituitary basophilism, or Cushing's disease. This is occasionally associated with parathyroid hyperplasia and osteoporosis. The blood chemistry is essentially normal in this condition. There are also associated obesity, hypertension and amenorrhea.

According to certain observers,[30] thyrotoxicosis may cause an unusual depletion of the calcium stores of the body. These stores, being housed in bone trabeculae, produce when depleted a demonstrable osteoporosis. Symptoms referable to the bones, namely, fracture and change in shape, may occur. The blood calcium values are normal, though increased amounts of calcium are found in the urine and feces. The basal metabolic rate is, of course, increased.

Not infrequently, osteitis fibrosa cystica is associated with one or more hyperostoses of the skull. Such a condition must be differentiated from true leontiasis ossea faciei of Virchow, osteoma, Hand-Christian-Schüller disease and giant cell or other tumors of the bone.

True leontiasis ossea has its onset in childhood, rarely progressing much after maturity is reached. It is confined solely to the skull.

---

28. Bentzon, P. K. G.: Roentgenological and Experimental Studies on the Pathogenesis of Dyschondroplasia (Ollier's Disease), Acta radiol. **3**:89-112, 1924.

29. Mahorner, H. R.: Dyschondroplasia, J. Pediat. **10**:1-26 (Jan.) 1937.

30. Williams, R. H., and Morgan, H. J.: Thyrotoxic Osteoporosis, Internat. Clin. **2**:48-60 (June) 1940. Bauer, W.; Aub, J. C., and Albright, F.: Studies of Calcium and Phosphorus Metabolism: Study of Bone Trabeculae as Readily Available Reserve Supply of Calcium, J. Exper. Med. **49**:145-161 (Jan.) 1929. Albright, F.; Bauer, W.; Cockrill, J. R., and Ellsworth, R.: Studies on Physiology of Parathyroid Glands: Relation of Serum Calcium to Serum Phosphorus at Different Levels of Parathyroid Activity, J. Clin. Investigation **9**:649-677 (Feb.) 1931. Aub, J. C.; Bauer, W.; Heath, C., and Ropes, M.: Studies of Calcium and Phosphorus Metabolism: Effects of Thyroid Hormone and Thyroid Disease, ibid. **7**:97-137 (April) 1929. Albright, F.; Bauer, W., and Aub, J. C.: Studies of Calcium and Phosphorus Metabolism: Influence of Thyroid Gland and Parathyroid Hormone upon Total Acid-Base Metabolism, ibid. **10**:187-219 (April) 1931.

According to Lederer,[31] who quoted Mayer,[32] the characteristic roentgen signs are as follows:

1. There are considerable thickening and increased density of the bone, the thickening being intense and uniform. 2. Surfaces of the affected bone are entirely smooth and sharply defined. 3. The disease does not go beyond the anatomic margins of the bone or of a part of the bone if the latter has developed from one or more bony "anlagen."

Pathologically, the changes are those of extreme osteoblastic activity with resulting replacement of bone by compact, eburnated osseous tissue, while the changes noted in osteitis fibrosa cystica are those of osteoclastic activity with very little if any osseous tissue present in the lesion. The blood chemistry in true leontiasis ossea is normal.

According to Ewing,[33] osteoma is the term applied to true neoplasms. It is very difficult to differentiate the true neoplasm from simple hyperplastic osseous growth secondary to trauma or infection. Ewing stated:

Spontaneous or traumatic but noninflammatory origin, progressive course, circumscribed form, active participation of osteoblasts, and derivation from cartilage are features which are more prominent in true osteoma, while an inflammatory origin, self-limitation, multiplicity, diffuse form, reduced numbers or absence of osteoblasts, and origin from connective or other soft tissues belong to the less definite of spurious bony neoplasms.

The absence of generalized skeletal changes as well as a normal blood chemistry easily differentiate this condition from osteitis fibrosa cystica.

Hand-Christian-Schüller disease resembles osteitis fibrosa cystica particularly when in addition to the deformity of the head there occur changes in the long bones with spontaneous fracture. This form of the disease is also known as Chester's disease. Pathologically, the characteristic foam cell composed chiefly of cholesterol esters is diagnostic. The Christian syndrome, i. e., defects in membraneous bones, exophthalmos and diabetes insipidus, as well as the increased blood lipids, including cholesterol, with all other constituents normal, is characteristic.

Other tumors about the head may be differentiated by examination of tissue.

Table 1 is a résumé of the features which might assist in the diagnosis of these various osseous diseases.

---

31. Lederer, F. L.: Idiopathic Hyperostosis of the Skull, Arch. Otolaryng. **34**:88-98 (July) 1941.

32. Mayer, E. G.: Ueber Röntgenbefund bei retrobulbären Erkrankungen unklarer Aetiologie und ihre differential diagnostische Wertung, Acta radiol. **9**:383-398, 1928.

33. Ewing, J.: Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1929, p. 213.

Disease	Location	Associated Constitutional Derangement	Roentgen Findings	Age-Sex Group	Pathologic Changes	Chemistry
Osteitis fibrosa cystica	Generalized and bilateral	Parathyroid tumor; pain in bones and muscles; fractures	Granular osteoporosis; multiple fractures; cortical expansion	All ages; usually females	Lacunar resorption with fibrous replacement of normal bone; cysts present	Increased serum calcium; increased serum phosphatase; decreased phosphorus; negative calcium and phosphorus balance
Polyostotic fibrous dysplasia	Generalized but unilateral	Pain in bones and muscles; fractures	Osteoporosis; multiple fractures; cortical expansion	Begins in childhood	Lacunar resorption with replacement by fibrous tissue	Normal except for increased phosphatase
Osteodystrophia fibrosa	Generalized and bilateral	Precocious puberty; pigmentation of skin; increased basal metabolic rate	Osteoporosis; multiple fractures	Starts before puberty	Lacunar resorption with replacement by cystic and fibrous tissue	High cholesterol, blood fat and blood sugar; normal calcium and phosphorus; phosphatase elevated
Osteitis deformans ("Paget's disease")	Generalized and bilateral	Weakness and heart disease	Coarse mottling with accentuation of the trabeculae; hyperostosis and sclerosis	Middle life	Invasion of bone by connective tissue; narrowing of marrow with thickening of cortex	Normal except for increased serum phosphatase
Osteomalacia (adult rickets)	Generalized and bilateral	Vitamin D deficiency; undernourishment	Osteoporosis; involvement of epiphysial ends of long bones	Child-bearing period usually	Softening of bone with formation of cysts	Low serum calcium and phosphorus with increase in phosphatase
Renal osteitis fibrosa cystica (rickets)	Generalized and bilateral	Marked renal insufficiency; enlarged parathyroids; hypertension	Osteoporosis; mottling or woolly appearance; calcification of small and medium-sized arteries	Usually adults	Lacunar resorption with replacement by fibrous cystic tissue; hydronephrosis and metastatic arteriosclerosis	Marked renal insufficiency; high nonprotein nitrogen; reduction of serum calcium with a decided phosphorus retention
Ollier's disease (dyschondroplasia)	Symmetric, involving diaphyses; may be unilateral (Ollier's disease) or bilateral (dyschondroplasia)	None	Longitudinal striations and gouged-out areas in diaphyses of long bones	Begins in childhood	Cartilaginous areas in shafts of long bones; secondary calcum deposits	Normal
Osteogenesis imperfecta	Generalized and bilateral	Blue sclerotics; deafness and relaxation of ligaments	Nodular areas of increased density; streaks of condensation along shaft	All ages	Deficiency in calcification; diminished trabeculae; medulla fibrous or lymphoid	Normal except for increased phosphatase
Thyrotoxicosis	Generalized	Increased basal metabolic rate; tremor, etc.	Osteoporosis	Adults usually; both sexes	Depleted calcium from osseous trabeculae	Calcium normal in blood but increased in urine and feces
Leontiasis ossea	Confined to skull	None	Thickened, eburnated bone	Starts in childhood; both sexes	Extreme osteoblastic activity with elimination of osseus tissue	Normal
Pituitary basophilism	Generalized	Obesity; hypertension and amenorrhea	Osteoporosis	Starts in childhood	Parathyroid hyperplasia; lacunar resorption	Normal
Osteoma of skull	Confined to skull	None	Circumscribed eburnated bone	All ages	Hyperplastic osseous growth	Normal
Xanthomatosis	Generalized, but particularly head	Defects in membranous bones, exophthalmos and diabetes insipidus	Localized rarefaction	Childhood usually	Characteristic foam cells due to deposition of cholesterol esters	Increased blood lipids, including cholesterol
Multiple myeloma	Generalized	None	Bone appears moth-eaten	All ages	Proliferation of some one cell of the marrow	Bence Jones protein present; serum calcium elevated
Bone metastases from malignant tumors	Generalized	Primary malignant growth	Extreme porosis	Any age, usually adults	Lacunar absorption with replacement by mother tumor tissue	Generally normal; serum calcium may be elevated; serum phosphatase usually increased

A report of a case follows. Of prime interest is the ease with which isografts take in these patients, even in close proximity to an infected cavity like the mouth.

REPORT OF A CASE

The case in question is one of osteitis fibrosa cystica occurring in a woman. Because of pain in the left maxillary area, associated with pain throughout the skeletal system, she was rather loosely classified as a "rheumatic" and advised to have her teeth extracted. This operation

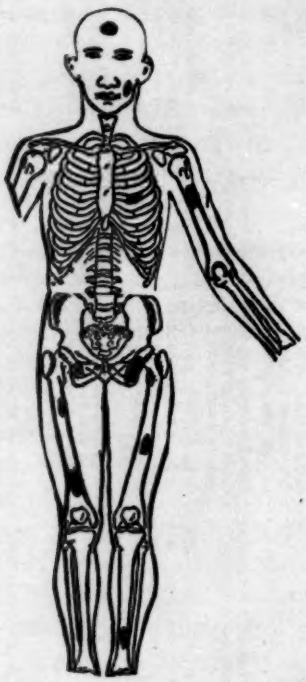


Diagram showing distribution of lesions.

resulted in a fracture of the upper jaw. Since only dental roentgenograms had been taken, a cyst involving the maxilla and palate had been overlooked.

B. G., a white woman aged 35, was referred for investigation of the sinuses as a possible etiologic factor of her rheumatism. She gave a history of pain in the regions of the left maxilla, right femur, left side of the chest and lower part of the left tibia. The pain began six years previously in the left facial area and spread gradually to the other involved parts. A year prior to examination she had had all her teeth extracted. In the process of extraction of an upper left third molar, the jaw was fractured. The gingiva healed quickly without secondary infection, but with considerable shortening of the jaw and deformity of the face. There was no history of fractures elsewhere in the body. Nothing significant was

elicited about her past or family history. Her birth and childhood were normal. No other members of her family suffered from any osseous disease.

General physical examination revealed little of note except painful areas over the lower end of the right femur, the left side of the chest at about the level of the sixth rib near its union with the sternum, and the lower end of the left tibia. There was considerable shortening of the left side of the maxilla. The left side of the hard palate was raised about 1.5 cm. above the level of the right, and much of its bone was absent, only a small amount being present in the midline, where it articulated with the palatine bone of the opposite side. Roentgen examination revealed decalcification with complete absence of bony structure of most of the body of the left maxilla and the left palatine bone. A similar area was noted in the right side of the frontal bone. This area, about the size of a dollar, showed an entire loss of all bony anatomic markings. Roentgen examination of the rest

TABLE 2.—*Laboratory Findings*

	Sept. 14, 1941	Dec. 12, 1941
Wassermann reaction	Negative	
Calcium	15.9 mg.	11.3
Phosphorus	2.8 mg.	3.6
Phosphatase	5.6 units	
Calcium balance	Negative	Ingested 400 mg. Excreted 1,240 mg.
Total proteins	8.71%	
Serum albumin	6.55%	
Serum globulin	2.16%	
Creatinine	1.6 mg.	
Nonprotein nitrogen	6.6 mg.	
Urea nitrogen	12.8 mg.	
Uric acid	2.5 mg.	
Cholesterol	154 mg.	
Sugar	108 mg.	
Total fatty acids	320 mg.	
Basal metabolic rate	—7	
Urinalysis	Normal	
Red blood cells	4,420,000	
White blood cells	16,200	
Differential white blood count	Normal	
Hemoglobin	90%	
Phenolsulfonphthalein test; total dye excreted in 2 hours	35%	

of the body revealed a similar area in the left sixth rib, one in the lower part of the left tibial and one in the inferior ramus of the left ischium. There were two such areas in the right femur, one about the size of a half-dollar at the lower end and one about the size of a quarter just cephalad to it. Other smaller foci were noted in the left femur, and left humerus (figure). Examinations in the laboratory gave the results presented in table 2.

Examination of the neck failed to reveal any palpable tumor.

Exposure of the lesion in the frontal bone revealed a solid, gritty, gray fibrous mass, which cut easily. No cystic material appeared to be present. Histologically the normal bone was entirely replaced by a rather avascular fibrous tissue. There were interlacing whorls of collagen fibers through which were interspersed numerous giant cells and fibroblasts of various ages. There was a notable absence of all osseous architecture, and no cartilage or osteoid tissue was present. Very few osteoblasts were noted in the areas of absorption.

Because of the poor condition of the patient, an exploratory operation on the neck was delayed several months. In the interim fractures of the left humerus and

right femur occurred. Eventually, an exploratory operation on the neck was accomplished and a parathyroid adenoma was removed. Within two weeks after this surgical intervention, the calcium and phosphorus values dropped nearly to normal.

Treatment.—The treatment of osteitis fibrosa cystica is directed primarily toward removal of the parathyroid tumor and secondarily toward prevention and repair of deformities due to fracture. Curettement of cysts before fracture and filling of the cavity with particles of bone has been the treatment of choice in the past.[34]

The excellent results of implantation of cartilaginous isografts, as well as the ease with which material for grafting may be obtained, have suggested the use of this method of repair, particularly about the bones of the head. This procedure was carried out to repair the facial deformity resulting from a telescoping of the maxillary bone. The involved area was curetted, the impaction separated and an isograft, 1 by ¼ inch (2.5 by 0.6 cm.) inserted in a previously prepared groove. In spite of the close proximity of the operative field to the mouth, the graft remained healthy and the normal facial contour was restored.

SUMMARY

Skeletal deformities about the head are frequently first seen by the otolaryngologist The case reported is one of osteitis fibrosa cystica. It was so diagnosed because of a characteristic roentgen and pathologic picture, a high calcium and a low phosphorus level in the blood, a negative calcium balance and a parathyroid tumor. Removal of the parathyroid tumor resulted in a return of the calcium and phosphorus levels, as well as of the calcium balance, to normal. The cyst in the face was curetted and filled with a cartilaginous isograft, the normal facial contour thus being restored.

841 East Sixty-Third Street.

34. Swift, W. E., and Hallock, H.: Treatment of Localized Fibrocystic Cavities in Bone by Curettage and Packing with Bone Chips, J. Bone & Joint Surg. **20**:411-418 (April) 1938.

CONTROL OF HEAD NOISES

THEIR ILLUSIONS OF LOUDNESS AND OF TIMBRE

EDMUND PRINCE FOWLER, M.D.

NEW YORK

If one disregards the auditory hallucinations occurring in organic diseases of the brain and in the psychoses, there are two kinds of tinnitus: (1) vibratory, caused by actual autogenous vibrations reaching the ear from any part of the body, and (2) nonvibratory, caused by biochemical irritation of the auditory neural mechanism.[1] Either of these two kinds of tinnitus may be superimposed on the other, in which case either one with sufficient intensity and proper frequency can diminish or increase the loudness and change the timbre and therefore the degree of annoyance of the other.

It is important to recognize these distinctions, because successful treatment of the disorders causing tinnitus, like successful treatment of the diseases or disorders causing deafness, depends on an understanding of the various factors involved. It is clear that any treatment that claims to be just for tinnitus or deafness is on its face unscientific and that no informed physician should be guilty of encouraging any such questionable procedures.

Limitations of time and space compel me to exclude at this time a full discussion of vibratory tinnitus. Moreover, vibratory tinnitus presents few difficulties in differential diagnosis or treatment as compared with the nonvibratory auditory sensations, because its physical characteristics are usually easily recognized, are often audible to the examiner and respond informatively to many physical manipulations. There is no primary illusion of sound or of timbre, because the tinnitus is a real sound. The loudness is dependent on the amplitude reaching the inner ear and on the degree and type of deafness. The annoyance depends largely on the psychologic reactions of the patient. Sensations caused by actual sound, odors, light, taste and movement are not usually exaggerated. They are received in accustomed amounts and natural combinations and can therefore be readily rationalized. They are real. There is no illusion.

Read at the Forty-Eighth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., June 1, 1942.

1. Fowler, E. P.: Head Noises and Deafness: Peripheral and Central, Laryngoscope **49**:1011 (Oct.) 1939; Tinnitus Aurium in the Light of Recent Research, Ann. Otol., Rhin. & Laryng. **50**:139 (March) 1941.

On the other hand, sensations caused by biochemical reactions or irritations of any of the nerves of sense are not caused by actual sound, light, odors, tastes or touch or actual movement of the body. They are unrelated to former experiences, in quantity and quality. They seem unnatural, and they are unnatural because they are an illusion and, like all illusions, tend to be exaggerated.

CONDITIONS COMMONLY ASSOCIATED WITH THE ILLUSION OF SOUND CALLED NONVIBRATORY TINNITUS

1. Impacted Cerumen.—This, depending largely on its amount and consistency and the pressure exerted on the drum, causes positive or negative pressure in the external auditory meatus and by way of the drum and ossicles can change the tension in the tympanic and labyrinth window tissues. It can by these means interfere with the normal aeration of the middle ear and its surrounding air spaces. Any of these factors, as well as prolonged paralysis or contractions of the stapedius and tensor tympani muscles, can change the circulation in the vessels of small caliber near the windows and in the adjacent walls of the labyrinth. It is only a fraction of a millimeter from the base of the basilar membrane to the round window and little more to the oval window, so that the high tone areas are easily brought into a changed milieu by vascular changes in or near either of the cochlear windows.

Vibratory tinnitus is frequently experienced from fluttering or tetanic contractions of the muscles of the middle ear. It is interesting to note that after severance of the tendon of the tensor tympani muscle, removal of the malleus or destruction of the drum membrane, all of which occur in radical mastoidectomies, tinnitus is usually absent or slight. Also during chronic suppurative otitis media there is usually no tinnitus.

2. Pathologic Closure of the Eustachian Tube.—This causes a partial vacuum in the middle ear and contiguous spaces by absorption of the trapped air and results in congestion of the vessels which anastomose with those in the bony capsule of the labyrinth (Pollitzer). The extent of the effect on the cochlear circulation depends largely on the design of the vascular tree.

3. Acoustic Trauma.—Few healthy American children grow up without having suffered some acoustic shocks, for instance from diving, explosions or blows on the ear. The drum membrane may not have furnished sufficient protection. People with perforations in the membrane from childhood are comparatively free from tinnitus. I believe this is due not only to the lack of imbalance of air pressures in the middle and the external ear when the tympanic membrane is perforated but to the lessening of trauma, because of the absence of an intact membrane, and to the protective barrier of the obstructive lesions which

always accompany such perforations. However, obstructive deafness renders the environment comparatively quiet, so that even faint tinnitus is easily sensed.

Faint tinnitus, and nonvibratory tinnitus generally, is not noticed until thrust on the consciousness after a traumatic lesion or one which suddenly disturbs the vascular and lymph balances of the inner ear. In this connection it should be kept in mind that it is not pressure which traumatizes or tears the cochlear structures but the excessive shearing action due to unequal pressures on the two sides of the basilar membrane or contiguous tissues. The greater the obstructive lesions the less the access for acoustic shearing pressures and the less the damage. The greatest effects from acoustic shocks are felt near the basal third of the cochlea, because it receives the greatest impact from the blow and because here the basilar membrane is narrower and the neural elements are acoustically more complex and sensitive and less mobile than in its distal two thirds. Acoustic shock is just as truly an actual physical trauma to the minute vessels and nerve elements in the cochlea as a hammer blow is to the minute vessels and nerves in a finger. It causes relatively as much, or even more, posttraumatic hemorrhage, edema, anoxia and local metabolic dysfunction.

After a severe acoustic shock there is tinnitus, with deafness for different sounds (sometimes for all sounds) of varying degree and duration, depending on the severity, position and extent of the lesions. There are a local concussion, microscopic petechial hemorrhages and maybe what can be called a resulting "cochlear apoplexy," the neural explosions being detectable only as tinnitus. If the damage is not too great, recovery takes place in inverse order to the severity of the lesion. Very frequently there is only a partial recovery. Narrow troughs, sometimes less than an octave wide, in the audiometric curve are seen between 2000 and 8000 double vibrations, but commonly there is no complete recovery for any of the tones above 3000 double vibrations (or lower), so that a marked dip then remains in the upper half of the audiogram threshold curve.

Trauma localized to the contents of the cochlea, unless severe or long continued, does not directly affect the auditory neural mechanism central to the spiral ganglion so that even though there is monaural total deafness the central auditory mechanism may still function, and being freed from normal stimulation may in some respects be even more sensitive to reflex intracranial disturbances than before. This may account for the occurrence in some persons of tinnitus referred to an ear with total deafness. Vascular changes by way of the autonomic nervous system will then be more free to irritate and so cause tinnitus, which in these

instances is usually an over-all frequency sound, like that of air or steam escaping simultaneously from nozzles of various sizes (it sounds like a thermal noise).

I am aware of the general opinion that the auditory nerve is not irritable to stimulation central to the end organ, but I do not subscribe to this opinion either as to the cochlear or as to the vestibular portion of the eighth nerve. All neurologists will aver that a first symptom of tumor of the cerebellopontile angle is tinnitus. I know of no reason to suppose that the auditory nerve is different from other nerves in its reaction to stimuli. As with any nerve, irritative phenomena may mean it is in the process of being destroyed.[2]

4. Otitis Media and Otosclerosis.—When these conditions sufficiently disturb the normal functioning of the cochlea windows, they result in some interference with movement of blood and lymph. Whether this results in tinnitus depends on several anatomic and psychoneurologic factors. The presence and loudness of the tinnitus from such stasis depend largely on its speed of onset, character and degree. Frequently there is little or no tinnitus. It is interesting to note that a fistula operation usually quickly stops the tinnitus, at least temporarily.

5. Drugs and Poisons.—Tobacco, quinine and the salicylates and their derivatives, excessive salt, coffee, tea, alcohol, carbon monoxide, carbon disulfide, arsenic, lead, phosphorus, mercury, morphine, chemotherapeutic drugs like aniline dyes and the sulfonamide and other compounds and many other poisons in sufficient amount will irritate, and some in sufficient concentration and with continued administration will destroy, the neural elements in the cochlea, the hair cells and the spiral ganglions and their neurons. Until the destruction is complete severe tinnitus usually results. Massive doses are seldom administered to human beings, but strong continued sublethal doses have been known finally to destroy in part or in toto the cochlear neural mechanism in susceptible persons. Smoking is a common cause of tinnitus. It should be stopped for at least a month to eliminate it as a factor.

6. Cardiovascular Disorders.—Intracranial arteriosclerosis, aneurysms, variations in blood pressure, internal paling and flushings and autonomic imbalance, as well as the lesions accompanying an idiopathic Ménière complex, anemia and old age, are very common causes of tinnitus. Their locations and therefore their effects are spotty. These disorders are best handled in cooperation with an internist, who will prescribe or guide the treatment indicated. Increased vascular pressure communicated to the cranial contents, such as that from a tight neck band, straining at stool or bending low, commonly causes tinnitus in older persons, but not in young persons with ample suppleness in muscles, joints and vascular channels.

2. A constant tinnitus long endured surely threatens an increasing and permanent nerve deafness.

7. Gastrointestinal Disorders.—Intestinal toxemia and gallbladder and gastric disturbances are often sufficient to set up or contribute to the determining factor in tinnitus. They should be studied and treated by a competent internist. As a simple measure, a glass of boiling hot water sipped slowly an hour before meals has often proved of great service.

8. Psychoneurologic Disturbances.—Apprehension, fright, loss of sleep, auditory and body tire, maladjustments in business and family life and depression often set up the mechanism for hair trigger firing of the auditory neurons. Although these disorders are in the field of the psychiatrist, he will usually prefer to turn them over to the otologist, who by proper understanding can usually best manage the aural symptoms. Sedatives in management of these conditions are useful.

9. Allergy.—So-called allergic reactions are not confined to the epidermal surfaces of the body. Their effects may be far reaching, even to the intracranial vessels and so to the lymph spaces in the cranial bones and to the neural mechanisms of hearing. Allergy may, and often does, cause irritation of the ear (tinnitus) as well as irritation (itching) in the nose or other sense organs. All patients with tinnitus should be questioned as to an allergic history. If there is such a history, a complete examination for allergy should be made. If a specific antigen is not found, histamine desensitization gives some promise in allergic tinnitus.

10. Idiosyncrasy.—The vascular tree in the auditory labyrinth varies among persons and even in the two ears of any person. This is also true of the vascular tree in the eye. It is one reason why ears vary greatly in their liability to the reactions causing tinnitus and deafness (also probably otosclerotic lesions).

11. Involvement of Otic, Gasserian and Geniculate Ganglions.—These ganglions are often implicated in the causation of tinnitus. They are very complicated and present almost a virgin field for research. The new sympathetic depressors, such as syntropan, are sometimes of use. In this connection one should consider temporomandibular and other disorders of the jaw and bony and other growths and infections about the head.

12. Intracranial Growths.—Because of the wide scattering and duplication of the acoustic fibers in the brain there may be no hearing defect from a tumor in the brain stem. Dandy stated that even a two-thirds division of the cochlear nerve may cause little or no loss of hearing and that the loss is restricted to the high tones.[3] Guild[5] reported limited

3. Dandy, W. E.: Partial Section of the Eighth Nerve, Tr, Am. Otol. Soc. **27**:393, 1937.

4. Footnote deleted by author.

5. Guild, S. R.: Studies on Human Temporal Bones: I. Discussion from the Point of View of Human Temporal Bones, Tr. Am. Otol. Soc. **25**:37, 1935.

change in the hearing following extensive cutting or destruction of the eighth nerve.

Disease does not show such clearcut results because of the scatter or mass patterns of some lesions. In this way one can account for many of the variations in sensation observed clinically. The rate of the abnormal tissue growth is important, because so long as there is no direct pressure there is time for equalization and adjustment to the crowding. There is then no destructive shearing stress. Even a large growth may be free from auditory effects. However, a gradually increasing tinnitus is an important symptom in the diagnosis.

COMMENT

It will be noted that all of these conditions associated with nonvibratory tinnitus seem to act directly or indirectly on the blood supply to the auditory neural elements or their supporting tissues. When they act indirectly it is commonly by way of the autonomic nervous system.

Nonvibratory tinnitus in my opinion may be brought about locally by several factors, namely, anemia, edema, diminished nutritional supply or inefficient removal of tissue wastes, stretching or tearing of nerve elements, local microscopic petechial hemorrhages, disturbances of pressure or water balance, affecting the movability or specific gravity of the intracranial fluids, changes in p_H and toxemia from infections or poisons of any kind. It will be noted that all of these incidentally produced anoxia, or lack of oxygen in the tissues, and therefore an edema and impending degeneration of the neural elements in the order of their sensitiveness to the poisoning or starving factors encountered. This is, of course, a simplified picture but quite comprehensive in its implications.

It is obvious that tinnitus from any cause is best treated by removing the cause, but this is often impossible, because not only does nonvibratory tinnitus vary on such slight alterations in the local environment of the ear and even of the body that the cause is not detectable, but the lesion is often permanent. The neural elements remaining under constant stress or irritation then usually continue to produce the tinnitus intermittently or constantly.

In many persons, no matter how loud it seems, tinnitus is accepted as a finality and simply allowed to buzz along in its accustomed way. Some tend to underestimate rather than overestimate the loudness of the tinnitus and the disagreeableness of its timbre. It may be sensed as a pleasing sound and even enjoyed.

On the other hand, there are persons who, because the tinnitus is not a pure tone, experience an exaggerated sensation as to both its loudness and its timbre, and it is then overestimated and sensed as a most disagreeable or unbearable noise.

The degree of exaggeration can be determined to some extent by measuring the loudness and timbre of the tinnitus. This is accomplished by having the patient compare the loudness of his tinnitus with the loudness of a similar sound applied to his opposite ear or by having him compare the tinnitus with some other sound, preferably not far distant from the tinnitus in frequency and intensity. The number of decibels over threshold required to balance the tinnitus in loudness indicates just how loud the tinnitus really is. This is an important step in the approach to diagnosis and treatment.

It is frequently observed that though a patient may say his noises are driving him crazy, or that they sound like a boiler factory, etc., they may in fact be very faint, commonly only 5 or 10 decibels above threshold. They are seldom more than 40 decibels above threshold. City dwellers are almost constantly in the presence of noise louder than this. These facts, which he himself has brought out by his own volition, must be firmly planted in his consciousness until he understands that they do not correspond to his statements as to the severity of the symptom. This is common sense psychology. The patient must be educated to rationalize his symptoms and accept them at their face value and not allow them to "get on his nerves."

In the treatment of tinnitus brought on by autonomic nervous system episodes, I have often found the following treatment of benefit, particularly in cases in which an attack of marked lowering of the hearing occurs with or immediately following ringing in the ears.

The patient is given 2 or 3 tablets of glyceryl trinitrate ($\frac{1}{100}$ grain [0.65 mg.]), with the instruction to take 1 immediately when he senses the aura or actually hears the tinnitus returning or increasing in volume. He is told that this will stop it, as fortunately it frequently does. Whether or not the result is due entirely to the glyceryl trinitrate (or similar drug), I believe that the mere fact of having, so to speak, an anchor to windward to stop the drift into the tinnitus and deafness is psychologically important. Every attack that is aborted tends to lessen the frequency or the severity of recurrences. At any rate, I have several young patients whom I now see but seldom because through several years they have promptly avoided or controlled their attacks in this way. Before I used this simple method, in spite of all I could do they often went for weeks before their tinnitus disappeared and their hearing returned to its former level. I believe that in similar cases this medicament or a similar-acting drug should be used. I have seen this type of deafness remain greatly increased or become total when the episodes of tinnitus and deafness were not controlled.

Tinnitus is common in older persons with cardiovascular disorders, and treatment along similar lines is therefore often indicated, because of course it may often benefit these disorders as well as the tinnitus.

When it is impossible to remove the tinnitus it is the duty of the otologist to lessen its annoyance, and this he can do by rehabilitation of his patient to a more normal mental and social status. To this end lip reading and hearing aids may be valuable. All these measures serve to lessen not only the tinnitus but frequently the deafness.

I have not dwelt on the differential diagnosis and the many phases of local treatment, which of course are important, because I wish to emphasize the method of approach more than the definitive treatment for each condition. I also wish to call attention to the conception that tinnitus is something more than a disorder of hearing.

140 East Fifty-Fourth Street.

TUBERCULOSIS AND THE OTOLARYNGOLOGIST

WILLIAM F. HULSE, M.D.
CLEVELAND

Otolaryngologists are frequently called on to manage the common cold and in so doing occasionally see the diagnosis of this condition undergo a startling metamorphosis. The alteration is usually in the direction of an acute contagious disease. A diagnosis of coryza often obscures a more important background, the serious nature of which may not be suspected for a week or ten days. The exact turning point at which significant differences occur is seldom sudden or obvious to the extent that patients outside the scope of hospital observation are sometimes separated from their original diagnosis at a time when adequate therapeutic measures are instituted too late for maximum benefits.

There are roughly two types of pulmonary tuberculosis—the "wet" and the "dry" type. The latter type may be in a rather advanced stage without producing suspicion. The former, usually displaying signs and symptoms which will eventually arouse suspicion, has an onset which is more often than not described in terms which fit perfectly the common cold. Many facts and much speculation about the common cold have been put on record, yet today it is shrouded in about as much diagnostic and therapeutic confusion as it ever was. One of the oldest maxims in medicine is that the common cold lasts two weeks. In most cases this is probably true if complications are not encountered. A short prodromal stage is almost always experienced, during which the mucous membranes are dry and easily irritated. The patient at this time has a feeling of undue fatigue and lassitude, and this is shortly followed by a period of organization during which the secretions become mucoid and purulent to varying degrees. This is the final stage and persists for five to ten days, during which time the secretions gradually subside. It is in the latter stage that careful observation is most indicated if any disease other than coryza is suspected. Detailed histories of patients in an institution for the treatment of tuberculosis will reveal many whose story of the onset of their disease is much like that just outlined. I do not imply that the common cold is a precursor of pulmonary tuberculosis but that the onsets are so similar that diagnostic confusion may easily follow. Where elaborate diagnostic methods are not available, there are no therapeutic tests which will easily separate one condition from the other. Various tests for specific contagious diseases which are at hand give

From the Sunny Acres Tuberculosis Sanatorium.

testimony in positive results with negative histories that the contagious diseases may brush the patients lightly in passing and leave for the records the fact that these patients have probably experienced just another cold. This is particularly true in cases of tuberculosis, in which positive tuberculin tests give striking testimony that most persons have had this disease before the first two decades of life have elapsed without presenting clinical evidence of its presence.

HISTORY

In any case in which tuberculosis is suspected a careful history may be relatively unimportant, as may also be the physical examination. Of patients with pulmonary tuberculosis in an early stage, there is an appreciable number whose physical examinations may give essentially negative results even in experienced hands. The demonstration of a positive contact, however, is definitely of importance and should be placed in a prominent position in the history. Most patients who are subjected to rather frequent colds will give a history for one which is usually like all the others if the cold is uncomplicated; any deviation should be carefully weighed. By far the greatest number of patients who have colds do not consult a physician unless complications relative to the ear, the nose or the throat have occurred or unless the cold seems to them to be running a protracted course. It follows that any patient presenting himself with a cold whose symptoms are not sharply localized to one or more of the upper respiratory pockets and whose cold has run an extended course should be carefully investigated. It is not meant by this that every patient who falls into this category should have a diagnosis of tuberculosis insinuated, but since about 2 of 3 adult persons in this country have had this disease in one degree or another, it may not be too remote a possibility that one may detect tuberculosis in patients of the type described, at its onset.

TEMPERATURE

Certainly no physical examination is complete without a reading of the patient's temperature. The common cold, if uncomplicated, is not characterized by whatever fever, if any, accompanies it. If bronchitis is present, the temperature may show a slight increase, but this seldom persists for more than a week. Acute otitis media or sinusitis is usually clearcut and may be an obvious reason for a rise in temperature. Any increase in temperature due to causes other than those mentioned should direct attention to either the pulmonary or the urinary system; this is particularly true in the examination of younger patients. A single reading of temperature is of little or no importance. Hospital records point out the obvious major role of the temperature curve, and yet most

private patients do not have the benefit of this type of observation unless a nurse is in charge. The untreated patient with pulmonary tuberculosis in an early stage has a rather characteristic temperature curve, low in the morning and high in the afternoon. If the duration of the cold is over two weeks, and the elevation in temperature still persists, the patient certainly deserves a careful thoracic examination. A single examination of the chest in the early stage of pulmonary tuberculosis may be of no value even in the hands of one thoroughly familiar with its routine. The classic signs and symptoms usually attributed to pulmonary tuberculosis should be kept in mind as belonging to moderately or far advanced disease and their limited presence in the early stage should be more clearly understood. In other words, those whose signs and symptoms are minimal or who have no signs cannot be given a clean bill of health until all the diagnostic measures are employed which may be deemed necessary. A carefully kept temperature chart may be one's most important clue to an early stage of tuberculosis.

URINE ANALYSIS

The kidneys have long been appreciated as indicators of disease elsewhere in the body when they themselves are not the primary seat of the difficulty. Their role in the diagnosis of tuberculosis may be of some importance. Since this disease is one which is characterized by the toxic products of its causative organisms one finds a renal response in an appreciable number of cases in that a trace of albumin is found with no evidence of renal damage. If the pulmonary changes are extensive, this may persist for some time, but in the early stage this soon disappears. Alone, certainly, this finding is relatively unimportant in the diagnosis of tuberculosis, but coupled with other facts it may be helpful.

MUCOUS MEMBRANES

Any acute or chronic respiratory disease affects to some degree the remainder of the respiratory system. The lesions may be concentrated in the lower region, but the remainder will not escape some manifestation. The substance secreted or excreted by tubercle bacilli, designated as tuberculin, has long been known to produce in every case of infection some degree of somatic allergic response to its presence. Since this allergic status originates in the respiratory system, the mucous membranes of this system will present some degree of allergic response at one time or another. In the early stages of pulmonary tuberculosis there will be a mucous membrane which is often intensely injected and somewhat edematous at times. After the disease has become established, the mucous membrane takes on an entirely different appearance. At this time I am not sure whether the appearance of the interior of the

nose is due to an unusual allergic response or whether it is due to the debilitating nature of the disease, but it is rather characteristic. The picture is one of atrophy, and this may be rather marked in cases of advanced disease. This is always accompanied by dryness and some degree of crusting. A small piece of mucous membrane was removed in a number of cases in which death from pulmonary tuberculosis had occurred. This material was removed from the same portion of the nasal chamber in all cases and showed microscopically marked atrophy. Patients with marked deviation of the nasal septum encountered in this institution seldom present the complications which are found in non-tuberculous persons with the same nasal picture. One would also imagine that extensive pulmonary suppuration would frequently be reflected in the upper air passages in some degree of suppuration, since the lining of the entire respiratory system is uninterrupted, but this is almost never found except as a terminal condition.

THE LARYNX

The larynx is not infrequently a reliable indicator of pulmonary involvement. Apical tuberculosis, especially that on the right side, occasionally produces hoarseness owing to its being adjacent to the recurrent nerve. The recurrent nerves may also become involved in mediastinal shift, which is found at times in pulmonary tuberculosis and which exerts torsion or tension on these nerves. This type of mechanical difficulty leads to weakness or paralysis of one or both vocal cords. In this clinic paresis of the vocal cord is not uncommon but is less likely to appear in the early stages. Many patients with early pulmonary tuberculosis present themselves with hoarseness due to coughing, profuse bronchial secretions, irritation or any combination of these factors. It should be pointed out that most of the patients with pulmonary tuberculosis who are hoarse do not have specific disease of the larynx. Tuberculosis of the larynx is not characteristic of early or minimal pulmonary tuberculosis, but hoarseness from indirect causes is met with often. Most cases of hoarseness unfortunately come under that wide and embracing category of laryngitis whose application has too often camouflaged something more grave, either locally or in the chest. The classic rule that a patient with a condition diagnosed as simple laryngitis must be given two weeks in which to get well seems still to be a rather important one to remember.

At this point it may be stated that laryngeal tuberculosis does not occur if the lung fields are clear. A few isolated instances have been reported refuting this statement, but most laryngologists feel that this condition does not occur alone. It is entirely possible, however, for laryngeal tuberculosis to occur in the early stages of the pulmonary

disease, and this fact should be kept in mind even though the probability is remote. In the greatest majority of the cases, early involvement of the larynx occurs in association with the hopeless or overwhelming type of disease in the chest, and this type leaves no doubt in the mind of the clinician as to the state of affairs.

RALES

Patients who visit the otolaryngologist are usually not the recipients of a complete physical examination; most men in this branch of medicine have no doubt lost their ability to carry out a complete examination because of this fact. One of the most important factors to be noted in an examination of the chest is the alteration of the breath sounds in abnormal pulmonary conditions. Probably the most important finding is the presence or the absence of rales. These are universally described as being heard with a stethoscope through the chest wall. I maintain that they may be distinctly heard and identified with the naked ear in a great many instances by listening to the patient breathe deeply through the mouth.

SUMMARY

The points mentioned in this paper are to be in no sense construed as final criteria in the diagnosis of tuberculosis but are presented as a collective suggestion that the otolaryngologist is in a more advantageous position to diagnose pulmonary tuberculosis than would be immediately apparent. Roentgen examination of the chest has not been mentioned in this paper; it is assumed that any one familar with the points stressed in this paper will know instinctively which way to turn.

EFFECT OF CIGARET SMOKE ON THE p_H OF THE THROAT

NOAH D. FABRICANT, M.D.
CHICAGO

Despite the fact that the annual consumption of cigarets by the American public has been continually increasing, little substantial scientific research on the effect of cigaret smoke on the upper respiratory tract of normal persons has been recorded in the medical literature.

Recently, the role of the hygroscopic agent—glycerin or diethylene glycol—in irritation of the respiratory tract has received some interest. Various approaches to the problem have been used in the published reports.

Mulinos and Osborne [1] and Haag [2] studied the edema-producing properties of smoke solutions in the conjunctival sacs of rabbits. Flinn [3] and Ballenger and Johnson [4] made clinical observations on irritation of the pharyngeal mucous membranes. Holck and Carlson [5] used the salivary response as their index of irritation. All these investigators used glycerin-treated cigarets and diethylene glycol–treated cigarets.

EXPERIMENTAL INVESTIGATION

In this study I have utilized a silver–silver chloride glass electrode in conjunction with the Coleman electrometer for the purpose of measuring the effect of cigaret smoke on the normal, physiologic range of the p_H of the mucous membrane of the human throat. In essence, studies of the p_H of the mucous membrane of the human throat apply a laboratory procedure in a clinical environ. The subjects were 100 men and women with clinically normal throats.

This investigation was aided by a grant from the Medical Relations Division, Camel Cigarettes.

1. Mulinos, M. G., and Osborne, R. L.: Pharmacology of Inflammation: III. Influence of Hygroscopic Agents on Irritation from Cigarette Smoke, Proc. Soc. Exper. Biol. & Med. **32**:241, 1934; Irritating Properties of Cigarette Smoke as Influenced by Hygroscopic Agents, New York State J. Med. **35**:590, 1935.

2. Haag, H. B.: Studies on the Physiologic Action of Di Ethylene Glycol, J. Lab. & Clin. Med. **22**:341, 1937.

3. Flinn, F. B.: Some Clinical Observations on the Influence of Certain Hygroscopic Agents in Cigarettes, Laryngoscope **45**:149, 1935.

4. Ballenger, H. C., and Johnson, V. H.: Effects on the Throat and Conjunctiva of the Hygroscopic Agent Used in Cigarets, Arch. Otolaryng. **25**:75 (Jan.) 1937.

5. Holck, H. G. O., and Carlson, A. J.: Alleged Influence of Glycerine and Diethylene Glycol upon the Irritating Qualities of Cigarette Smoke, Proc. Soc. Exper. Biol. & Med. **36**:302, 1937.

Data on One Hundred Subjects Used in Experiments on the Effect on the p_H of the Throat of Smoking Cigarets

Patient No.	Sex	Smoking Habits	p_H Range
1	M	½ package daily	5.70-6.21
2	M	1¼ packages daily	5.85-6.30
3	M	Occasional cigaret	6.60-7.55
4	F	1 package daily	6.03-6.70
5	M	1½ packages daily	6.07-6.70
6	M	Cigars only	6.42-7.00
7	F	½ package daily	5.65-6.30
8	F	10 cigarets daily	6.20-6.66
9	F	1½ packages daily	5.89-6.70
10	M	2 packages daily	5.79-6.31
11	M	1 package daily	5.71-6.11
12	F	6 cigarets daily	6.12-6.72
13	F	½ package daily	6.62-7.44
14	M	10 to 15 cigarets daily	6.50-7.07
15	M	Does not smoke habitually	6.81-7.35
16	F	Does not smoke habitually	7.41-8.00
17	M	Smokes a pipe chiefly	6.35-7.65
18	M	Smokes occasionally	5.40-6.71
19	F	Does not smoke habitually	6.87-7.47
20	F	½ package daily	6.60-7.18
21	F	1 package daily	6.98-7.62
22	F	½ package daily	5.90-6.33
23	F	½ package daily	6.52-7.54
24	M	1½ packages daily	6.55-7.40
25	F	1 package daily	5.73-6.24
26	F	1 package daily	5.87-6.23
27	F	1½ packages daily	7.11-7.85
28	F	1½ packages daily	5.82-6.48
29	M	2½ packages daily	6.56-6.81
30	M	1½ packages daily	6.10-7.27
31	M	Does not smoke habitually	6.74-7.42
32	M	1 package daily	5.20-6.07
33	F	Does not smoke habitually	6.96-7.52
34	F	Smokes occasionally	7.00-7.90
35	M	A pipe daily	4.93-6.80
36	M	1 package daily	6.35-7.00
37	F	1 package daily	6.10-6.80
38	M	Does not smoke habitually	6.22-7.12
39	F	Does not smoke habitually	6.70-7.22
40	M	1 package daily	6.42-8.00
41	F	Does not smoke habitually	6.73-7.64
42	F	½ package daily	6.33-7.20
43	M	1 package daily	6.31-6.92
44	F	1 package daily	4.98-6.64
45	M	1 package per week	6.98-7.56
46	M	1 package daily	6.90-7.31
47	M	1 package daily	5.94-6.38
48	F	1 package daily	5.90-6.54
49	M	½ package daily	6.82-7.45
50	F	½ package daily	5.80-6.52
51	F	½ package daily	6.75-7.50
52	M	1 package daily	6.45-7.82
53	M	1¼ packages daily	5.40-6.69
54	F	¾ package daily	4.91-6.37
55	M	1½ packages daily	5.30-6.65
56	M	1 package daily	4.90-5.55
57	M	A pipe and cigars	6.87-7.62
58	M	1 package daily	5.70-6.88
59	M	1 package daily	5.45-7.10
60	M	1½ packages daily	4.92-6.13
61	F	Does not smoke	6.81-7.28
62	F	Smokes occasionally	6.01-6.62
63	M	Smokes occasionally	5.72-6.38
64	F	1 package daily	5.40-6.21
65	M	1 package every other day	6.00-7.34
66	M	Does not smoke	6.60-7.36
67	F	Smokes occasionally	6.50-6.81
68	F	Smokes occasionally	6.56-7.35
69	M	Does not smoke	7.09-7.75
70	M	1½ packages daily	6.04-6.46
71	M	Rarely smokes	6.54-7.31

Data on One Hundred Subjects Used in Experiments on the Effect on the p_H of the Throat of Smoking Cigarets—Continued

Patient No.	Sex	Smoking Habits	p_H Range
72	M	1 package daily	5.84-6.20
73	F	1½ packages daily	6.38-7.15
74	M	1 package daily	7.04-7.50
75	M	Smokes occasionally	6.18-6.83
76	M	Smokes occasionally	7.21-7.73
77	M	Does not smoke	6.40-7.69
78	M	Does not smoke	6.75-7.25
79	F	1 package daily	6.13-6.62
80	M	Rarely smokes	6.86-7.75
81	F	½ package daily	6.32-6.73
82	M	2 packages daily	6.87-7.48
83	F	Does not smoke	6.10-6.65
84	F	10 cigarets daily	6.19-6.66
85	F	6 cigarets daily	6.31-7.08
86	F	8 cigarets daily	6.31-7.25
87	M	1 package daily	5.60-6.60
88	M	Smokes occasionally	6.30-6.90
89	M	1 package daily	6.15-6.71
90	F	2 packages daily	5.63-6.11
91	F	1 package daily	5.69-6.01
92	F	Smokes occasionally	6.25-6.95
93	M	1 package daily	5.65-6.40
94	F	1 package daily	5.60-6.00
95	M	Smokes pipe daily	6.89-7.19
96	F	Smokes occasionally	6.18-6.59
97	F	Smokes occasionally	6.09-6.50
98	F	1 package daily	7.00-7.32
99	M	½ dozen cigars daily	6.42-6.92
100	F	½ package daily	5.67-6.30

Elsewhere [6] I have described the use of the silver–silver chloride glass electrode in conjunction with the Coleman electrometer for the determination of the nasal p_H in health and in disease. I have also reported that the normal, physiologic range of the p_H of the throat in 51 men and women with clinically normal throats varied from 4.9 to 8.0, the reaction in the majority of cases falling in the acid range. The various precautions necessary to attain accuracy are dealt with in the published communications.

The method used in this experiment is simple. The glass electrode is inserted into the patient's mouth so that it is brought into contact with the mucous membrane of the supratonsillar region immediately adjacent to the upper pole of the tonsil. At this site, the contact of a glass electrode can be tolerated by most patients. Placing the glass electrode directly on the mucous membrane of the postpharyngeal wall is unsatisfactory, for the pharyngeal reflex immediately comes into play and interferes with the accuracy of the reading.

Unlike the method utilized for the determination of the nasal p_H, it was found that in the throat the glass electrode cannot remain in situ for long periods. The glass electrode is inserted; a reading of the p_H is taken, and then the electrode is withdrawn after each reading. Minute to minute readings were made during the entire experimental period. The futility of relying on only one p_H reading is

6. Fabricant, N. D.: Significance of the p_H of Nasal Secretions in Situ, Arch. Otolaryng. **34**:150 (July) 1941; Further Studies: Significance of the p_H of Nasal Secretions in Situ, ibid. **34**:297 (Aug.) 1941; Effect of Silver Preparations and Antiseptics on the p_H of Nasal Secretions in Situ, ibid. **34**:302 (Aug.) 1941; An Introduction to the p_H of the Throat, ibid. **37**:169 (Feb.) 1943.

illustrated by the frequent changes in values that occur when readings are recorded for any appreciable length of time.

Three kinds of cigarets were used in the experiments: the first, a cigaret using glycerin as the hygroscopic agent (Camels); the second, a cigaret using diethylene glycol, and the third, a cigaret which was specially made so as to contain no hygroscopic agent. The glycerin-treated cigarets and the diethylene glycol–treated cigarets were purchased at random; the cigarets without a hygroscopic agent were made to conform in weight and all other physical characteristics to the purchased cigarets.

In the conduct of the experiments the speed and volume of the puff, the frequency and number of puffs and the position of the cigaret while burning were left entirely to each smoker's preference and habits. Some of the smokers discarded a cigaret after a half-dozen puffs; the majority, however, discarded it much later. While some inhaled deeply, others did not. Each smoker was cognizant of the type of cigaret he was smoking. No attempt was made to alter in any way a smoking technic which may be called the average smoking habit.

At the beginning of each experiment, ten readings of the p_H of the mucous membrane of the throat—one reading per minute—were taken to determine the normal, physiologic range of the p_H for that subject. He was then permitted to smoke as long as he wished and in strict accordance with his own smoking habits. In general, the interval between puffs for the majority of subjects varied from thirty seconds to one minute. A p_H reading was taken each minute between puffs. When the subject was ready to dispense with the cigaret he was permitted to do so. Minute to minute readings of the p_H were continued after the cessation of smoking in order to study the after-effects of the smoking and were terminated only by the time allotted for the experiment.

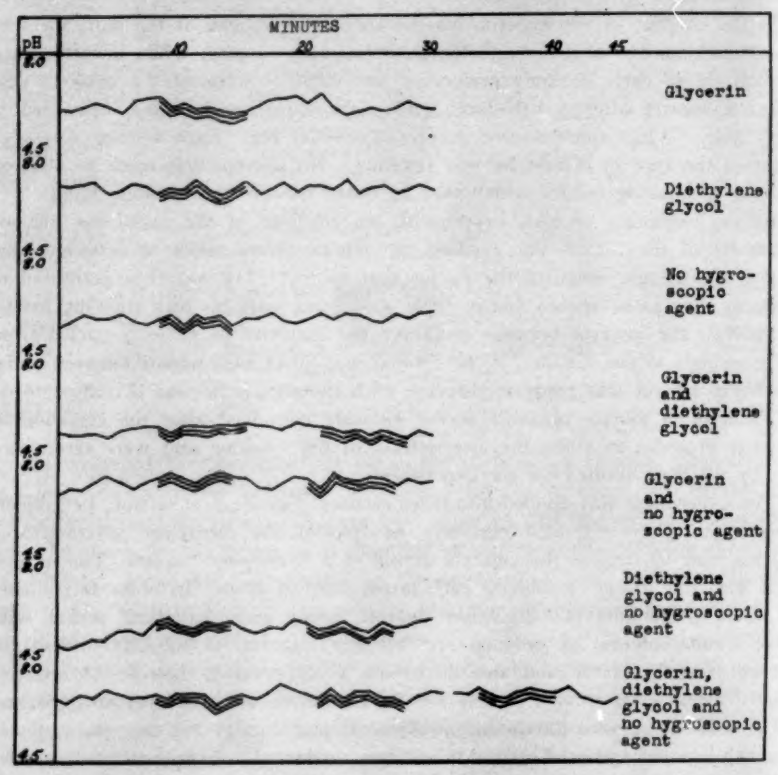
The experiment was divided into three sections. In the first section, 10 subjects smoked the glycerin-treated cigarets; 10 smoked the diethylene glycol–treated cigarets, and 10 smoked the cigarets devoid of a hygroscopic agent. The experiment with this group of subjects each lasted one-half hour. In order to compare the effect of smoking two dissimilar cigarets during a one-half hour period, with a six minute interval of smoking rest between cigarets, 15 subjects smoked the glycerin-treated cigaret and the diethylene glycol–treated cigaret; 15 subjects smoked the glycerin-treated cigaret and the cigaret devoid of a hygroscopic agent, and 15 subjects smoked the diethylene glycol–treated cigaret and the cigaret devoid of a hygroscopic agent. Finally, 25 subjects smoked the three cigarets—glycerin-treated, diethylene glycol–treated and hygroscopic agent-free—with six minute intervals of smoking rest between cigarets. In this group each individual experiment lasted forty-five minutes.

RESULTS

The table shows the sex of the subjects, their smoking habits and the range of the p_H of the mucous membrane of their throats: The one reported range indicates the values before, during and after smoking of the three kinds of cigarets, for no change from the normal was found to result from smoking. The accompanying figure contains representative graphs of the effect on the p_H of the throat produced by the three kinds of cigarets.

COMMENT

The importance of the relationship of the p_H to inflammation has been stressed both by Menkin [7] and by Hansel.[8] In his recently published textbook, "Dynamics of Inflammation," Menkin stated that the usual cellular sequence in the development of an acute inflammatory reaction consists in an initial infiltration of polymorphonuclear leukocytes, which



Graphs showing the effect on the p_H of the throat produced by three kinds of cigarets. The periods of smoking are indicated by triple lines.

are subsequently replaced by an abundance of macrophages (i. e., cells acting as scavengers when the inflammatory irritant has been overcome).

The correlation between the p_H and the cytological picture in inflammation suggests that the hydrogen ion concentration is the factor conditioning the cellular

7. Menkin, V.: Dynamics of Inflammation: An Inquiry into the Mechanism of Infectious Processes, New York, The Macmillan Company, 1940.

8. Hansel, F. K.: (*a*) Allergy of the Upper and Lower Respiratory Tracts in Children, Ann. Otol., Rhin. & Laryng. **49**:579, 1940; (*b*) Cytologic Observations on the Secretions of the Nose and Paranasal Sinuses in Allergy, Tr. Am. Laryng. A. **62**:292, 1940.

pattern of an exudate. In vitro studies add further support to the view that the viability of an inflamed area is a direct function of the p_H. The hydrogen ion concentration thus determines the survival of leucocytes.

In the field of allergy, Hansel [8] asserted that the cytologic content of nasal and sinal secretions may be considered an approximate index of the pathologic processes occurring in the tissues and that the cytologic observations give important diagnostic information. He found that repeated cytologic examinations of the secretions of the nose and paranasal sinuses as a diagnostic procedure and as a means of determining the clinical course with respect to complicating infections is of indispensable value. The eosinophil-neutrophil ratio is an index of the nature and stage of the infection.

Elsewhere Hansel declared:

In the acute cold the hydrogen ion concentration of the secretions and tissues is first on the alkaline side, then sharply shifts to the acid side when the resolution stage is reached. In allergy, it is particularly significant that when the p_H falls low on the acid side there is a complete disappearance of the eosinophiles. When the p_H returns to the alkaline side, there is a return of the eosinophiles. These studies are not only interesting but important. They show that physico-chemical and cellular reactions play an important part in allergy and in immunity to infections.

In view of these observations, namely, (1) that the hydrogen ion concentration determines the survival of leukocytes and (2) that the cytologic content bears a significant relationship to the p_H milieu in cases of nasal allergy, measurements of the p_H can be considered one of the ways of determining a normal and an abnormal physiologic state and irritation of the mucous membranes of the nose and throat. Minute to minute recordings of the p_H are feasible in human beings; an attempt to study the cytologic content of mucous membranes each minute for prolonged periods is neither feasible nor particularly informative.

By conducting minute to minute readings of the p_H values of the mucous membrane of the human throat, it is possible satisfactorily to show the effect of cigaret smoke on the p_H of the throat. It is significant that the normal, physiologic range of p_H values for the throat in men and women with clinically normal throats lies between 4.9 and 8.0. The 100 subjects undergoing p_H measurements in the series recorded were, for the most part, accustomed to smoking either habitually or occasionally; several persons were nonsmokers. Cigaret smoke, regardless of the absence or presence of a hygroscopic agent and of the kind of hygroscopic agent, in no way altered the normal, physiologic p_H range of their throats. Likewise, during the conduct of the experiments, no objective evidence of irritation was observed. In terms of effect on the p_H of the throat, it was impossible to differentiate between the smoker and the nonsmoker.

Although in this series of 100 subjects there was no incompatibility between the smoking of cigarets and the normal, physiologic p_H values for the throat, it is conceivable that a tobacco-sensitive or tobacco-allergic person or one with an acutely inflamed throat might show alterations in the p_H range.

The effect of smoking two, and even three, dissimilar cigarets within a prescribed period—at a rate faster than most persons smoke—indicated no abnormal alteration of the normal, physiologic p_H of the throat. Apparently, the cumulative effect of several cigarets was no different than the effect on the p_H produced by the smoking of a single cigaret. The smoking of a glycerin-treated cigaret did not disturb the normal, physiologic p_H range of the mucous membrane of the throat. A comparison of the effect of the glycerin-treated cigaret and that of the diethylene glycol–treated cigaret failed to show any difference between them. Further, there was no difference between the diethylene glycol–treated cigaret and the cigaret devoid of a hygroscopic agent, nor was there any difference between the effect of the glycerin-treated cigaret and that of the hygroscopic agent–free cigaret with respect to the normal, physiologic p_H of the human throat.

CONCLUSIONS

A method is described involving minute to minute readings of the p_H values of the mucous membranes of the human throat for studying the irritating effects of cigaret smoke.

The p_H range of the clinically normal throat was found to vary from 4.9 to 8.0, the reaction in the majority of cases falling within the acid range. While the values varied with different persons, for the same subject the range was small and fairly constant.

Cigaret smoke, regardless of the kind of cigaret, in no way altered the normal, physiologic p_H range of the throats of 100 men and women. In terms of the effect on the p_H of the throat, it is impossible to differentiate between the smoker and the nonsmoker.

It cannot be stated that the type of hygroscopic agent used in the manufacture of various brands of cigarets influences the irritating properties. None of the cigarets used in this experiment caused a significant change in the normal p_H range of pharyngeal tissues.

ABSCESS OF THE TEMPOROSPHENOID LOBE

A REVIEW, WITH REPORT OF A CASE COMPLICATED BY PETROSITIS

MAURICE D. GRANT, M.D.
NEW YORK

Although Hippocrates knew the dangers to the brain in aural disease, the association of aural suppuration with abscess of the brain was regarded for many centuries as purely accidental. Morgagni, in the eighteenth century, was one of the first to voice the opinion that aural suppuration was primary and abscess of the brain secondary. Previous to this, even Valsalva had regarded the abscess as primary. Morand,[1] in the "Opuscules de Chirurgie" published in Paris in 1768, as quoted by Scott, published the first case report of otitic abscess of the brain, with recovery after operation. In 1873 Luther Holden,[2] in the *St. Bartholomew's Hospital Reports,* recorded an operative recovery from otitic abscess of the brain. After this, Gowers and Barker,[3] Schondorff [4] Truckenbrod,[5] Schwartze [6] and Horsley [7] reported successful operations on the brain for abscess. In a classic monograph published in 1893, Macewen [8] reported 15 cases of abscess of the brain with 14 recoveries. It was this report, with its detailed accounts of each case and the treatment therein, that firmly established the necessity for surgical drainage in the treatment of abscess of the brain.

ETIOLOGY

In 1934 Courville and Nielsen [9] reviewed 147,604 autopsies from Continental European clinics, and found 277 cases of abscess of the

---

Read before the Bronx Otolaryngological Society, Sept. 23, 1941.

From the Otolaryngological Service of Dr. Goodlatte B. Gilmore, Morrisania City Hospital.

1. Morand, S. F.: Opuscules de chirurgie, Paris, G. Desprez & P. A. Le Prieur, 1768, p. 161; cited by Scott, S.: J. Laryng. & Otol. **50**:106, 1935.
2. Holden, L.: St. Barth. Hosp. Rep. **9**:96, 1873.
3. Gowers, W. R., and Barker, A. E.: Brit. M. J. **2**:1154, 1886.
4. Schondorff: Arch. f. klin. Chir. **31**:316, 1884-1885.
5. Truckenbrod, C.: Ztschr. f. Ohrenh. **15**:186, 1885-1886.
6. Schwartze, cited by Braun, E.: Arch. f. Ohrenh. **29**:163, 1890.
7. Horsley, V.: Tr. Clin. Soc., London **19**:290, 1886.
8. Macewen, W.: Pyogenic Infective Diseases of the Brain and Spinal Cord, Glasgow, J. Maclehose & Sons, 1893.
9. Courville, C. B., and Nielsen, J. M.: Acta oto-laryng. **21**:19, 1934.

brain, an incidence of 0.19 per cent. In 45,272 autopsies on patients with otitis media, acute or chronic, there were 68 cases of abscess of the brain, an incidence of 0.15 per cent. Courville and Nielsen,[9] in reporting on 1,225 such abscesses following otitis media, found that 80 per cent followed chronic otitis media and 20 per cent followed acute otitis. Two thirds of all the abscesses in this series were found in the cerebrum; 90 per cent were solitary and 10 per cent were multiple. Twelve per cent occurred in the first decade of life, 57 per cent in the second and third decades, 15 per cent in the fourth decade, 9 per cent in the fifth decade and 7 per cent in all those past 50 years.

PATHOGENESIS

Infection can reach the brain by one of three routes: (1) by direct implantation due to violence or surgical intervention, (2) by contiguous extension from an adjacent source of infection or (3) by metastatic extension from a nonadjacent source through the blood stream. Abscess of the brain most frequently develops by contiguous extension from an adjacent source, and otitis media is the focus in the great majority of cases. In a series of 194 abscesses of the brain reported on by Evans,[10] 123 (63 per cent) were due to contiguous extension from an adjacent focus, and of these, 109 (56 per cent of the total) were from the mastoid, 12 (6 per cent) were from the paranasal sinuses and 2 (1 per cent) were due to invasion of the skull by malignant disease.

In the spread of infection from the mastoid, the most frequent manifestation is necrosis or perforation of the tegmen tympani or tegmen antri with involvement of the overlying dura. However, infection may reach the dura by retrograde venous thrombosis, by dehiscences in the tegmen or through preformed pathways along nerve sheaths or foramens. After it reaches the dura, there are three routes by which the infection may invade the brain substance itself: (1) through the perivascular, or Virchow-Robin, spaces, (2) by retrograde venous thrombosis or (3) through the arteries. Macewen,[8] Eagleton,[11] Wittmaack[12] and Heine and Beck[13] have expressed the belief that retrograde venous thrombosis is the route of invasion. Atkinson[14] and Piquet[15] have

10. Evans, W.: Lancet **1**:1231, 1931.

11. Eagleton, W. P.: Brain Abscess, New York, The Macmillan Company, 1922.

12. Wittmaack, K., in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 12, p. 102.

13. Heine, B., and Beck, J., in Denker, A., and Kahler, O.: Handbuch der Hals-, Nasen- Ohrenheilkunde, Berlin, Julius Springer, 1927, vol. 8, pt. 3, p. 201.

14. Atkinson, M.: Ann. Otol., Rhin. & Laryng. **47**:1020, 1938.

15. Piquet, J.: Cong. franç. d. oto-rhino-laryng., 1935.

contended that the perivascular spaces form the most common route. According to both Eagleton and Macewen, the perivascular is a possible alternate route in selected cases.

The infection in traversing the subarachnoid space en route to the brain proper sets up a localized leptomeningitis with the formation of adhesions between the dura, the arachnoid and the pia, thereby sealing off the subarachnoid space at the point of invasion. This process and the retrograde venous thrombosis that proceeds to the subcortical area where the abscess develops is the abscess "with stalk" described by Koerner,[16] who found a "stalk" in 42 per cent of all contiguous abscesses of the brain. In a series of 75 abscesses examined both microscopically and macroscopically, Carmichael, Kernohan and Adson [17] could find no evidence of "stalk" formation. Yet Atkinson [14] stated that a point of entry can be found on the dura in every case of contiguous brain abscess. Evans [18] reported that in 43 cases out of a series of 74 there was an infection of the dura binding it down to the pia-arachnoid, and the abscess, from its macroscopic appearance, was separated from the meninges by a zone of unaltered cortex. The route of invasion in these cases was probably along the perivascular spaces of perforating blood vessels.

PATHOLOGY

The development of an abscess of the brain and the rapidity of its spread depend on the type and virulence of the invading organism and the resistance of the host. The abscess starts in the white matter just below the cortex as an area of localized encephalitis, the center of which breaks down and forms an abscess. Until recently it was believed that the arteries entering through the cortex and those entering the brain through the base were end arteries. However, the investigations of Pfeifer,[19] Cobb [20] and Lorente de Nó [21] have revealed a capillary network connecting the cortical and central arteries in these areas. The subcortical area, owing to its less efficient blood supply, is a relatively unprotected region in which infection may readily start.

Carmichael, Kernohan and Adson [17] described a completely formed abscess as having four layers: (1) a central necrotic zone made up of

16. Koerner, O.: Die otitischen Erkrankungen des Hirns, der Hirnhäute und der Blutleiter, ed. 2, Frankfurt, J. Alt, 1896.

17. Carmichael, F. A., Jr.; Kernohan, J. W., and Adson, A. W.: Histopathogenesis of Cerebral Abscess, Arch. Neurol. & Psychiat. **42**:1001 (Dec.) 1939.

18. Evans, W.: Lancet **1**:1289, 1931.

19. Pfeifer, R. A.: Die Angioarchitektonik der Grosshirnrinde, Berlin, Julius Springer, 1928.

20. Cobb, S., in Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932.

21. Lorente de Nó, R.: J. f. Psychol. u. Neurol. **35**:19, 1927.

leukocytes, lymphocytes, monocytes, gitter cells and cellular debris, (2) a vascular granulomatous border surrounding the central necrotic zone, (3) a zone of hyperemia and fibrosis and (4) an external layer of gliosis. Both Homén [22] and Alpers [23] described four layers, but Homén spoke of the fourth layer as a rarefied area composed of edematous brain tissue surrounding the abscess, and Alpers called it an encephalitic zone. The edematous or encephalitic zone may extend for a considerable area beyond the abscess proper and account for symptoms of pressure otherwise inexplicable.

The question of capsule formation is one of prime importance, since on it often rests the ultimate success or failure of surgical treatment of abscess of the brain. If one operates too early, before complete encapsulation has taken place, a spreading encephalitis is found and the infective process is diffused to other areas. If one waits too long, the patient may die suddenly owing to rupture of the abscess into the ventricle from an unexpected rise in intracranial pressure. Not all abscesses act alike in the development of a capsule. The time for encapsulation to occur and the type of capsule formed depend on the nature of the invading organism and the resistance of the host. Brunner [24] and Neumann [25] have pointed out that anaerobic bacteria, by their destructive effect, prevent formation of a capsule, whereas aerobic bacteria, especially the capsular cocci, produce a layer of fibrin which leads to the formation of a capsule.

Generally speaking, the older the abscess, the greater the likelihood of encapsulation. In Alpers' [23] series, a well defined capsule developed in the average case at the end of three weeks after intracranial invasion. He cited a case with beginning formation of a capsule after three days and a case in which there was a satisfactory capsule at the end of fourteen days. In 1 of his cases there was no evidence of formation of a capsule at the end of eleven months. If a capsule will form at all, it will be well defined at the end of three to six weeks.

The same organisms are found in a contiguous abscess of the brain as in the focus itself. Hemolytic streptococci and pneumococci are the organisms most frequently found in otitic and sinal abscesses. In metastatic and in post-traumatic abscesses the predominant invaders are staphylococci.

---

22. Homén, E. A.: Arch. a. d. path. Inst. d. Univ. Helsingfors **1**:1, 1913.

23. Alpers, B. J.: Abscess of Brain: Relation of Histologic to Clinical Features, Arch. Otolaryng. **29**:199 (Feb.) 1939.

24. Brunner, H.: Otogene endokranielle Erkrankungen, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 10, p. 194.

25. Neumann, H.: Otitic Cerebellar Abscess, London, H. K. Lewis, 1909.

SIGNS AND SYMPTOMS

In the initial stage or period of invasion, the signs are those of meningeal irritation. There may be a chill varying from a feeling of cold or chilly sensation to a violent paroxysm, slight fever, headache with neuralgic pain, nausea and vomiting. The initial chill, when it occurs, marks the time of intracranial invasion. In Yaskin's [26] series of 23 abscesses of the temporal lobe, a chill was not manifest in a single case, although it was present in every one of 17 cases of abscess of the frontal lobe. In the manifest stage there are signs of increased intracranial pressure accompanied by a dull, persistent headache and retarded mental processes. The temperature is normal or subnormal, and the pulse is slow. The temperature may have sudden rises followed by a fall to a subnormal state much like that seen in meningitis. In the latent stage there may be only a boring headache and mental dulness with a slight evening rise in temperature persisting for weeks.

The most notable and constant symptom is a continuous, deep and boring headache. It comes on early and may become intense, often being localized directly over the abscess or on the involved side of the head. Occasionally percussion of the skull reveals a tender area over the site of the abscess. More often, the headache is generalized over the entire head.

A retardation of the mental processes is typical. The patient appears less alert and is apathetic. Somnolence and restlessness may be present.

Vomiting may be an early or a late manifestation. When vomiting occurs in the early stages, it may be so conspicuous that it overshadows the underlying disease. In the late stages, vomiting is due to increased intracranial pressure and is projectile in type. Nausea is occasionally seen with the vomiting, and attacks of dizziness may be experienced, especially when the position of the head is changed or a sudden effort is made.

Changes in the eyegrounds are seen in about 60 per cent of cases. The manifestations vary from a passive hyperemia with dilated tortuous veins, which is common, to a marked choking of the disk, which is rare. When papilledema occurs, its subsidence after surgical drainage is looked for as an index of the diminution of the intracranial pressure. The prognosis with respect to the eyegrounds is good after successful surgical treatment.

The findings in the spinal fluid are variable, depending on the stage and progress of the disease. In the stage of invasion, there is a circumscribed leptomeningitis with an elevation of the spinal fluid pressure.

---

26. Yaskin, J. C.: Neurologic Complications of Infections of Temporal Bone and Paranasal Sinuses: Summary of Twenty Years' (1919 to 1938) Experience, Arch. Otolaryng. **30**:174 (Sept.) 1939.

Generally there are an increase in the cell count of 100 to 1,000 or more cells, predominantly polymorphonuclear leukocytes, and also an increase in the albumin and globulin content. After the initial invasion, the pressure falls to normal and the cell count becomes normal or is slightly elevated, the cells being mostly lymphocytes, but the albumin and the globulin remain increased. Indiscriminate spinal taps are decried, since they may cause hemorrhages into the meninges or a rupture of the abscess into the ventricle. However, when indicated, a careful aspiration with the removal of 1 or 2 cc. of fluid may be done without risk.

FOCAL SIGNS AND SYMPTOMS

The position of the primary focus is of the greatest importance in localization. Sixty-six per cent of all abscesses following disease of the mastoid are found in the adjacent temporal lobe, and 33 per cent are encountered in the ipsilateral cerebellar lobe. The two outstanding findings of localizing value in cases of abscess of the temporosphenoid lobe are aphasia and hemianopsia. The aphasia is usually sensory and is manifested by an inability to understand the meaning of the written or spoken word. It occurs only in left-sided lesions in right-handed persons and vice versa. Hemianopsia, when present, is a reliable sign. Changes in the visual fields are caused by pressure of the abscess on the optic radiations which traverse the inferior horn of the lateral ventricle on their way to the occipital lobe. The hemianopsia is on the contralateral side, right-sided hemianopsia indicating involvement of the left temporal lobe, and vice versa. The macular area is spared, so that there is no effect on central vision. A contralateral hemianopsia may be the only localizing sign in an abscess of the right temporal lobe in a right-handed person. Pressure from a large abscess may cause symptoms referable to remote regions of the brain. Thus a patient may present jacksonian seizures, contralateral central facial paralysis, hemiparesis on the same on opposite side, involvement of the trigeminal, abducens or oculomotor nerve and olfactory hallucinations. The most significant of all these signs is a contralateral central facial paralysis. This symptom in the presence of aural suppuration on the opposite side in a patient complaining of a dull headache who appears sullen and apathetic is characteristic of an abscess in the temporosphenoid lobe.

Roentgen studies are of limited value. Gas may form in an abscess and reveal its position, or the pineal body, if it can be visualized, will give valuable information. Air studies are infrequently reported in the literature. The danger of rupture of a thin-walled abscess during encephalography is always present. This type of examination might be tried in cases in which localization by all other means is impossible.

TREATMENT OF ABSCESS OF THE BRAIN

A number of principles have been established for the treatment of abscess of the brain and have been almost universally adopted. First and foremost, such abscess is not regarded as creating a surgical emergency, and a waiting period of from three to six weeks from the time of intracranial invasion is allowed for encapsulation and localization to take place. During this waiting period the primary focus is eradicated, and the patient is studied in order to localize accurately the site of the lesion. Before exploration, the dura over the site of the suspected abscess should be exposed in a circumscribed area which is isolated from the surrounding meninges by a gauze pack moistened in a mild antiseptic solution, or the periphery of the exposed dura should be sealed off by electrocoagulation. The wound is closed. After twenty-four to forty-eight hours the wound is reopened and the exploration for the abscess is made. This procedure is designed to close the subarachnoid spaces and prevent meningitis.

The site of operation is still a controversial subject. In general, otologists favor operating through the primary focus, whereas neurosurgeons prefer a clean field. Operations for abscess of the brain fall into two main groups depending on the size of the opening in the overlying bone. Coleman [27] and his followers believe that making of a small trephine opening and tapping, repeated if necessary, or tapping with the introduction of a small rubber catheter as a drain is sufficient. Otologists and a majority of neurosurgeons use a large opening in the bone, incise or excise the cortex down to the abscess wall, evacuate the pus by aspiration with a needle, then enlarge the opening in the abscess and either pack the cavity with iodoform gauze or rubber drains or leave it wide open and allow herniation.

Grant [28] in 1938 reviewed the later progress in 31 cases of recovery of three years or longer. Of 22 patients that had been treated with a small opening and tapping or tapping and drainage, 5 had neurologic sequelae that kept them from working and 17 had no demonstrable sequelae. Of 8 patients that had to be operated on through a large opening, 7 were incapable of resuming any gainful occupation.

Bucy [29] and Rowe [30] have shown that sulfanilamide and its derivatives are invaluable in the prophylaxis and treatment of the meningitis which formerly was the most frequent and most dreaded complication in brain abscess surgery. Chemotherapy, with full dosage, should be started on the day before operation and continued if a susceptible organism is found.

---

27. Coleman, C. C.: Treatment of Abscess of the Brain, Arch. Surg. **18**:100 (Jan., pt. 1) 1929.

28. Grant, F. C.: Prognosis for Abscess of the Brain, Arch. Neurol. & Psychiat. **40**:603 (Sept.) 1938.

29. Bucy, P. C.: Ann. Surg. **108**:961, 1938.

30. Rowe, S. N.: Ann. Surg. **107**:620, 1938.

PROGNOSIS

Since Macewen [8] reported 14 recoveries in 15 cases of surgically treated abscess of the brain, no one has reported an equal record. Grant [31] reviewed the operative mortality from abscess of the brain as reported from the leading clinics of the world, and found it to vary from 50 per cent to 75 per cent. At the present time, with the concept of therapy as outlined, he estimated that the mortality in selected cases should be 20 per cent and in all types of cases 40 per cent.

REPORT OF CASE

M. D., a white man aged 36, was admitted to the Morrisania City Hospital (otolaryngologic service of Dr. G. B. Gilmore) on April 18, 1940, complaining of pain in the right ear with discharge.

History.—The present illness had begun three weeks before as an acute infection of the upper respiratory tract accompanied with pain in the right ear. The pain in the ear had become worse and the drum had been incised two weeks before the patient's admission to the hospital. The ear had since been draining profusely. Three days before admission the patient complained of pain and headache localized on the right side of the head.

Physical Findings.—Examination revealed a thin, pale man, looking ill and as if in pain. The pupils were equal and regular and reacted both to light and in accommodation. The left ear was normal. The right ear had a profuse purulent exudate in the external canal. The canal was narrowed, and the drum was red, perforated and thickened. There were slight tenderness over the mastoid and subperiosteal edema. The peritonsillar area on the right side and both tonsils were injected. Examination of the heart, lungs, abdomen and extremities gave essentially normal results. The temperature on admission was 100.6 F., the pulse rate 76 and the respiratory rate 20. A blood count showed 11,800 white cells, with 87 per cent polymorphonuclear leukocytes and 13 per cent lymphocytes. The urine showed a heavy trace of albumin but no sugar. Microscopic examination revealed many epithelial cells, a few granular casts and some amorphous debris. There were 3 to 5 red blood cells, and 6 to 8 white blood cells, per high power field. The reaction to the Kahn test was negative. A roentgenogram taken on the day after admission showed an extensive destructive process in the right mastoid. No abnormality was seen in the left mastoid.

Operation.—The patient was operated on on April 20, 1940. A simple mastoidectomy was performed and revealed a necrotic, well pneumatized mastoid. The tegmen antri was entirely destroyed, and free pus was exuding from an opening on the under surface of the right temporosphenoid lobe of the brain. The opening, which was about 1 cm. in diameter, led into an abscess cavity which extended for a distance of 4 cm. into the substance of the brain. The postauricular incision was extended upward, the temporal muscle was split vertically, and with a rongeur a portion of the squamosa about 3 cm. in diameter was removed. This revealed the dura overlying the lateral surface of the brain. It was thickened, gray and apparently bound down to the pia-arachnoid. Three braided silkworm gut drains, which had been sewed together, were introduced

31. Grant, F. C.: Internat. Abstr. Surg. **72**:118, 1941; in Surg., Gynec. & Obst., February 1941.

into the abscess. This drain was sutured to the dura at the margins of the opening. The mastoid cavity was packed with iodoform gauze, and the cutaneous wound was left open.

Postoperative Course.—For the first two weeks the patient's postoperative course was uneventful. The wound was dressed daily, and the drain in the brain was left undisturbed. On May 4, the fourteenth postoperative day, the patient complained of headache, pain behind the right eye and in the right side of his head and double vision. Neurologic examination gave negative results, except for revealing a paralysis of the right external rectus muscle. There was no papilledema, and the visual fields in both eyes were normal. The temperature at this time was 99.8 F., but the pulse rate was only 50. Roentgen studies done on this day did not reveal any involvement of the petrous tip. On the sixteenth postoperative day, the patient was taken to the operating room for an inspection of the wound. As the drain was removed there followed a small amount of thick pus. A piece of rubber tubing was inserted into the cavity. On the seventeenth postoperative day the patient appeared better and was relieved of his headache. The palsy of the sixth cranial nerve persisted. On the nineteenth postoperative day a profuse purulent exudate was noted coming from the right external auditory canal. Roentgenographic examination of the petrous pyramids showed an increase in cloudiness of the right petrous apex with a suggestion of a destructive process at the tip. On the twenty-first postoperative day the patient again complained of a right-sided headache. The temperature at this time was 98 F., and the pulse rate was 58. The headache and slow pulse persisted, and the drain was removed on the twenty-fourth postoperative day.

Repeated neurologic examinations gave negative results except for showing the paralysis of the sixth nerve. Ophthalmologic investigation showed normal fundi, no defects in the visual fields and normal confrontation reactions. On the twenty-eighth postoperative day the patient felt better. His headache and pain had ceased, and the discharge from the external auditory canal, which had been profuse, had diminished. The palsy of the abducens nerve, however, was still present. A roentgenogram of the petrous pyramids taken on the thirty-first postoperative day showed the same cloudiness in the right petrous apex as did the previous roentgenogram. Cultures of material taken both from the abscess and from the mastoid at operation on April 20 grew hemolytic streptococci in broth. Cultures of material taken from the abscess on May 6, when the drain was replaced, grew hemolytic streptococci and Staphylococcus aureus in broth.

The discharge from the canal gradually diminished and ceased, and the postauricular wound completely healed. On June 3 the patient was discharged from the hospital, completely cured except for a palsy of the right external rectus muscle of moderate degree. By the end of June the palsy had completely disappeared, and the patient had perfect coordination of all the extraocular muscles. He has been in good health ever since and has no demonstrable sequelae at the time of writing.

COMMENT

This case illustrates the rapidity with which an otitic abscess of the brain can develop and become completely encapsulated. Without any history of infection prior to the present illness, a walled-off abscess was found three weeks after the onset of an acute infection of the upper respiratory tract and two weeks after myringotomy. The infection

proceeded through the tegmen antri, which was completely disintegrated, to the overlying dura and then by retrograde venous thrombosis to the brain. The actual time of intracranial invasion in this case is difficult to determine. However, it can be stated that a capsule delimiting the abscess developed in less than twenty-one days.

Except for a right-sided headache, which is as typical for acute mastoiditis as for abscess of the brain, there were no general or focal signs of an abscess previous to operation. The patient was right handed and consequently did not show any signs of aphasia. The abscess did not encroach on the optic radiations at the inferior horn of the lateral ventricle, and no changes in the visual fields occurred. There was no papilledema. The pulse corresponded with the temperature. There were no signs of increased intracranial pressure.

The development of a Gradenigo syndrome twenty-four days after operation is noteworthy. Friesner and Druss [32] expressed the belief that the Gradenigo syndrome is due to the meningeal reaction which ensues later in the course of a petrositis, rather than to the petrositis itself. This explanation is in agreement with that of Gradenigo himself, who ascribed the syndrome to a circumscribed leptomeningitis which encroaches on the abducens nerve as it passes through Dorello's canal and on the ophthalmic division of the trigeminal nerve as it passes over the anterior surface of the petrous apex. It must be remembered that a Gradenigo syndrome may be caused not only by petrositis but by an extradural abscess, a temporosphenoidal abscess, thrombosis of the lateral, superior petrosal or inferior petrosal sinus or meningitis. In this case, it might have been caused by pressure from the encephalitic zone surrounding the abscess itself or by a suppurative involvement of the petrous pyramid. The positive roentgen evidence and the profuse purulent discharge from the external auditory canal which appeared five days after the onset of the retro-orbital pain and palsy of the abducens nerve point to a diagnosis of acute suppurative petrositis. The discharge from the external canal would have been due to the formation of a fistulous tract from the petrous apex along the anterior cells of the petrous pyramid to the middle ear and the external auditory canal, contributing to the ultimate recovery of the patient.

Dr. Clarence H. Smith made suggestions that were used in the preparation of this paper.

1860 Morris Avenue.

32. Friesner, I., and Druss, J. G.: Osteitis of Petrous Pyramid of Temporal Bone, Associated with Paralysis of External Rectus, Arch. Otolaryng. **12**:342 (Sept.) 1930.

Case Reports

FOREIGN BODY IN THE BRONCHIAL TREE

Occurrence of an Object of Unusual Size

BERTRAM CAPUS, M.D., AND SOLOMON P. SCHECHTER, M.D., NEW YORK

Foreign bodies in the bronchi are not rare. A foreign body such as was found in this case usually lodges in the esophagus or the larynx rather than in the bronchus. This case is presented because of the following unusual features: the extraordinarily large size and the location of the foreign body; the asymptomatic course; the difficulty of roentgenographic visualization, and the mechanical difficulty of removal.

REPORT OF A CASE

E. W., a 60 year old man, presented himself to the otolaryngology outpatient department of the Metropolitan Hospital, with a history of having swallowed a beef bone three weeks ago. At that time he had a short coughing spell and a sharp pain in the left side of the chest. Since then he had complained of coughing spells associated with mild pains in the chest. Examination at the clinic of the hypopharynx and larynx revealed nothing abnormal. The patient was admitted to the Metropolitan Hospital for complete examination and bronchoscopy.

Physical examination showed no abnormalities. Fluoroscopy did not reveal an area of atelectasis or other evidence of the foreign body. A series of roentgenograms of the chest were taken; the posteroanterior view showed no abnormality (fig. 1 *A*), and the left oblique exposure revealed a suspicious faint shadow in the region of the bronchus of the lower lobe of the left lung (fig. 1 *B*), which was considered to be either a calcified lymph node or, possibly, a foreign body.

The following day bronchoscopy was performed, using a 9 mm. Jackson bronchoscope. The larynx and trachea were normal. The right main bronchus and its subdivisions and the left main bronchus and the bronchus of the upper lobe of the left lung were found to be normal. Just below the spur of the bronchus of the upper lobe of the left lung, within the bronchus of the lower lobe, a spur with an unusual whitish sheen was seen running parallel with the anterior surface of the body. Contact with the forceps revealed this to be the foreign body. There was adequate room around the foreign body for passage of air into the bronchus of the lower lobe of the left lung and sufficient room above and below to apply a foreign body forceps without injury to the bronchus. The foreign body was tightly wedged in the bronchus and could not be easily moved upward or downward or rotated in the bronchus with the forceps. After several attempts, it was possible slowly but forcibly to draw the foreign body up to the mouth of the bronchoscope. Because of its size, it was impossible to remove the body through the bronchoscope. Simultaneous removal of the bronchoscope and the forceps holding the foreign body was attempted, but the foreign body would not pass between

From the Department of Otolaryngology, Metropolitan Hospital; Dr. J. Hetrick, Director.

the cords because of its size and was lost from the grasp in the forceps. Reexploration revealed the foreign body in the right main bronchus at the level of the bronchus of the upper lobe, and removal was again attempted in a similar manner.

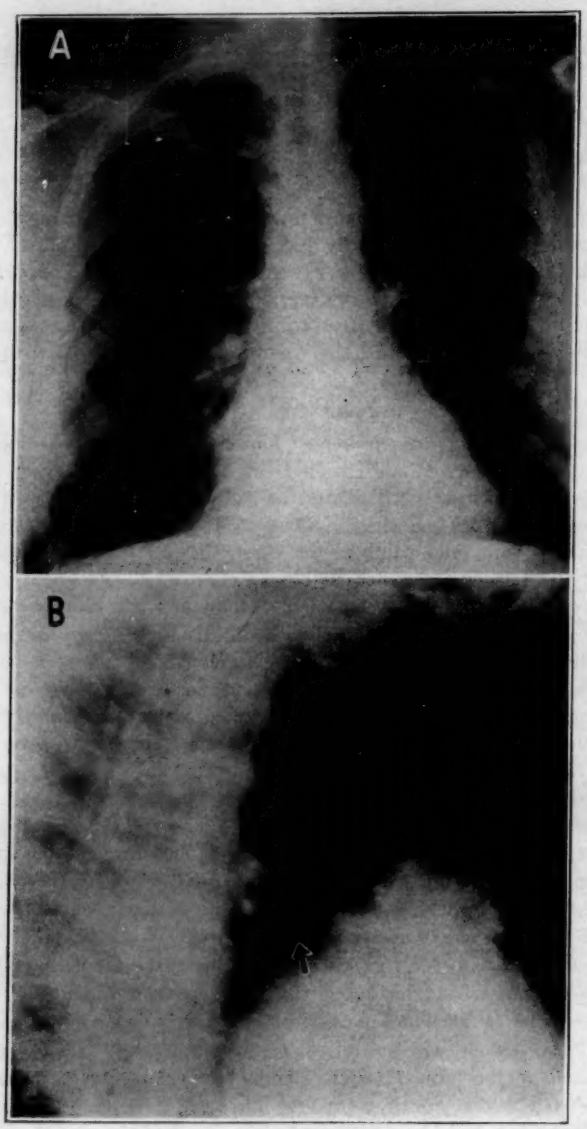


Fig. 1.—Posteroanterior (*A*) and left oblique (*B*) roentgenograms of the chest. In *B* the arrow indicates the foreign body.

At the vocal cords, once again, difficulty was experienced because of the size of the body, despite rotation to utilize the maximum diameter of the glottic space.

By exerting some slight force and bringing the foreign body as far posterior in the glottis as possible, removal was accomplished. Examination of the larynx after removal showed a small mucosal laceration in the interarytenoid region. The foreign body was a beef bone, which measured 7 by 12 by 23 mm. (fig. 2). The postoperative course was uneventful.

COMMENT

A brief consideration of the anatomic size of the bronchial tree and the larynx explains why this foreign body was unusual and its removal

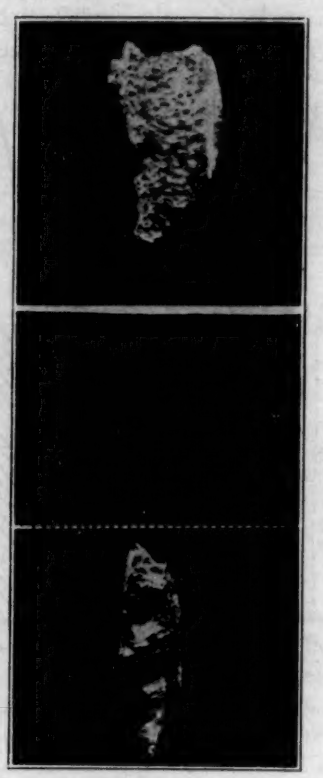


Fig. 2.—Photographs of the foreign body after removal.

difficult. Jackson [1] stated that in the adult man the glottic triangle measures 12 by 22 by 22 mm., thus enabling one to pass a 10 mm. bronchoscope. The diameters of the trachea are 14 by 20 mm. Piersol [2] stated that the diameter of the bronchus of the lower lobe of the left lung

1. Jackson, C., and Jackson, C. L.: Bronchoscopy, Esophagoscopy and Gastroscopy, ed. 3, Philadelphia, W. B. Saunders Company, 1934.

2. Piersol, G. A.: Human Anatomy, ed. 9, Philadelphia, J. B. Lippincott Company, 1930.

after the apical branch leaves it is 12 mm. Aeby [3] stated that the diameter of the left main bronchus just above the bronchus to the upper lobe is but 10.1 mm. and below it but 7.7 mm. The bronchoscopic observations in this case tend to corroborate Piersol's [2] figures.

Comparison of the diameter of the left main bronchus with the numerical measurements, 7 by 12 mm., of the foreign body in this case shows that the body could not descend lower than the level of the apical bronchus. The difficulty in passing a foreign body 7 by 12 mm. through the glottic triangle is easily explained by the geometric construction (fig. 3). From this figure it is realized that such a foreign

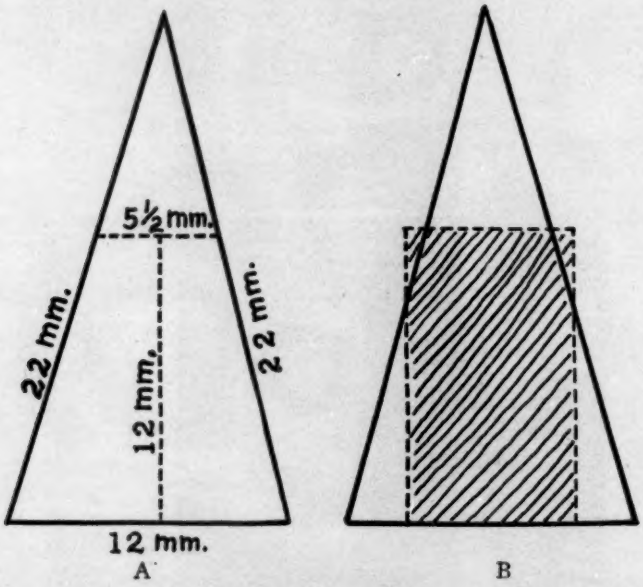


Fig. 3.—(*A*) A scale drawing of the larynx with a 12 mm. perpendicular erected on the base of the glottic triangle shows that the maximum diameter available 12 mm. from the base is 5.5 mm. (*B*) The blocked-out area represents a foreign body 7 by 12 mm., which is superimposed on the laryngeal triangle.

In this figure, 1 mm. represents ⅛ inch (3 + mm.).

body will barely pass through the glottic triangle if it is brought far posteriorly into this space and the vocal cords are stretched.

100 Van Cortlandt Park South.

1749 Grand Concourse.

3. Aeby, C. F.: Der Bronchialbaum der Säugethiere und des Menschen. Leipzig, W. Engelmann, 1880.

FIBROSARCOMA OF THE LARYNX IN AN INFANT

RALPH G. RIGBY, M.D., AND PAUL H. HOLINGER, M.D., CHICAGO

Malignant tumors of the larynx rarely occur in infants. They are of interest to the pediatrician and to the laryngologist alike because of the rapidity with which they may produce laryngeal obstruction. The following case is worthy of note because of the age of the infant, the type of tumor and its point of origin.

REPORT OF A CASE

R. E., a 17 day old white boy, was admitted to the Children's Memorial Hospital, Chicago, on June 23, 1941, because of a loud inspiratory crow and "blue" spells which had been present since birth. Oxygen had been administered at frequent intervals when the inspiratory crow persisted or was associated with the "blue" spells. These attacks occurred repeatedly when the infant cried or exerted himself. Roentgen treatments had been given for suspected enlargement of the thymus, but no improvement was observed.

Physical examination revealed that the infant was well developed and well nourished and weighed 8 pounds 4 ounces (3,855 Gm.). He did not appear acutely ill but had a definite inspiratory crow and difficulty in breathing. Dulness was found on percussion over the upper left side of the chest, being more pronounced anteriorly than posteriorly. Auscultation of the chest was unsatisfactory because of the loud inspiratory and expiratory laryngeal sounds, which were due to the laryngeal obstruction. A tentative diagnosis of congenital laryngeal stenosis complicated by partial collapse of the upper lobe of the left lung was made. Direct laryngoscopic examination carried out on the day of admission revealed that the left false cord and the left arytenoid cartilage were cystic and involved in a mass which almost completely obstructed the laryngeal airway.

During the next two weeks the physical signs in the chest cleared, but the inspiratory crow persisted and the patient required oxygen occasionally. Severe dyspnea made it necessary to supplement feeding by gavage to prevent exhaustion. Two weeks after admission to the hospital a second direct laryngoscopic examination confirmed the original impression that the left half of the larynx was involved in a cystic mass. Efforts to aspirate the contents with a needle were unsuccessful, and therefore a tracheotomy was performed in order to relieve the dyspnea. A no. 0 tube was used. A large vascular plexus was encountered overlying the trachea. Respiratory relief was immediate after the introduction of the tracheal airway, and the child began to gain weight steadily. Direct inspection of the larynx from time to time during the ensuing three weeks showed practically no change in the previously recorded picture.

After this period the airway through the larynx became completely obstructed, and the involved area was now observed to have a more firm and less cystic appearance. Another attempt, on August 1, to aspirate fluid from the left false cord failed. On August 4 an infant bronchoscope was passed through the larynx

From the Children's Memorial Hospital and the Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine.

into the trachea, and a granuloma, which had evidently been the source of blood in the aspirated mucus, was removed from the tracheotomy stoma. Histologic section showed it to consist of granulation tissue. The larynx was dilated each week during the following two months, and definite improvement in the airway was noted, the cords abducting more than at any previous time. On October 3 inspection of the larynx revealed that it opened sufficiently on inspiration to permit easy visualization of the vocal cords, and a no. 18 laryngeal bougie was introduced, without resistance to its passage. The dilation was repeated a week later, but the following day the mucus in the tracheobronchial tree began to increase in amount

Fig. 1.—Photograph of the specimen, showing the base of the tongue, the epiglottis and the posterior wall of the trachea, which has been opened. The tumor is visible in the larynx, protruding from the left.

and tenacity and was occasionally blood streaked. The infant had a few cyanotic spells during the following two days, in spite of the fact that the tracheotomy tube was patent. These attacks were relieved immediately by inhalations of oxygen. The temperature remained normal, as it had throughout the stay in the hospital. On October 16 the infant seemed to tire easily and became cyanotic at times. Humidifiers were employed to aid in liquefying the heavy mucus, and by midnight the amount of mucus decreased and was thinner. The child appeared to be breathing more easily and slept quietly. However, early the next morning, he

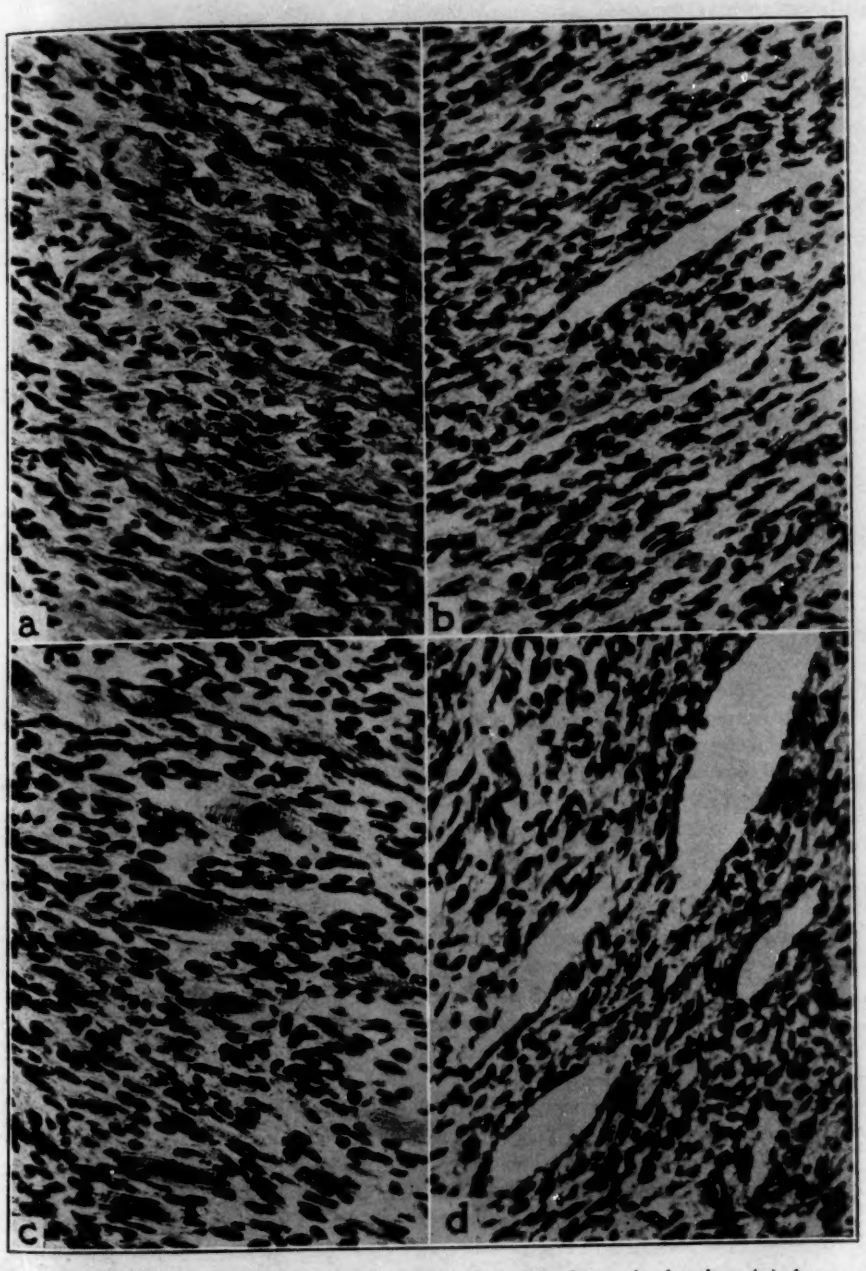


Fig. 2.—Photomicrographs of the tumor shown in figure 1, showing (*a*) large masses of elongated, spindle-shaped cells with hyperchromatic nuclei; (*b*) small, slitlike endothelium-lined channels in the tumor tissue; (*c*) outer margins of the large masses of spindle-shaped cells, with the striated muscle fibers interspersed, and (*d*) outer margins of the tumor, presenting large, slitlike endothelium-lined channels, a few of which contained blood cells.

began to have increased respiratory difficulty, and large amounts of mucus, much of it blood streaked, were aspirated through the tube. At 5:25 a. m. the child suddenly stopped breathing, and all efforts to revive him were unsuccessful.

Autopsy.—A complete postmortem examination, with the exception of the intracranial contents, was made.[1] Only the larynx and its adjacent structures are described in detail in this report because the lesion in question was limited to these areas. The blood culture revealed the presence of Staphylococcus aureus haemolyticus. Cardiac hypertrophy, myocardial edema and bilateral pulmonary edema were present.

A large tumor was observed on the left side of the larynx protruding into the lumen of the larynx and extending along its left lateral margin. The growth was continuous with a mass of enlarged, firm lymph nodes, 3 or 5 mm. in diameter, lying along the left side of the trachea. The trachea was opened along its posterior side below the larynx and a small amount of thin, blood-stained mucus was observed in the lower ends of the main bronchi and in the larger bronchioles of both lungs. No pathologic condition was evident in the lower bronchi to account for the origin of this bright red blood.

The tumor was well circumscribed and measured 1.5 by 2 by 3 cm. in diameter. It had pushed aside the thyrohyoid ligament, and the mass bulged into the lumen of the vestibule of the larynx, almost completely obliterating it. The cuneiform and corniculate cartilages were forced backward and to the right, the left horn of the corniculate cartilage lying to the right of the midline. The aryepiglottic fold was flattened and raised over the tumor, but the left ventricle was not obliterated. The left vocal cord appeared unaffected. On cross section, the laryngeal structures were observed to be pushed ahead of the mass but were not infiltrated by it. The tumor was composed of firm, white, glistening tissue of homogeneous appearance, without a definite capsule. Immediately adjacent to it was a small, irregular, nodular mass, measuring 7 by 13 mm., which appeared grossly to consist of enlarged, confluent lymph nodes.

Histologically, only the deeper layers of the submucosa were invaded by tumor tissue. Where cartilage was present, the perichondrium had not been invaded. The main portion of the tumor consisted of large masses of elongated, spindle-shaped cells with hyperchromatic nuclei of a similar form. There was a tendency for the cells in some areas to be arranged in parallel rows, while in others small whorls were formed. In more hyperchromatic areas the nuclei were somewhat irregularly shaped. One could search many fields before finding mitotic figures, but in others they were frequently seen. The tissue contained many small, slitlike, endothelium-lined channels which lay directly on tumor tissue. A few of these contained blood cells. There was slight tendency toward the deposition of collagen, but the small amount present was observed on fascial margins where nerve trunks had been compressed. At the outer margins of the large masses of spindle-shaped cells the tissue was interspersed with striated muscle fibers, and finger-like projections of spindle-shaped cells projected into the muscle bundles. The muscle fibers were mature, with distinct striations. At the periphery a few large, multinucleated muscle fibers were seen, which were probably proliferating sarcolemma sheaths or fibers that had been undergoing pressure atrophy. The histologic picture was that of a fibrosarcoma.

---

1. The postmortem examination and histologic studies were carried out by Dr. William G. Hibbs and Dr. Marion Barnes, of the Children's Memorial Hospital.

COMMENT

Numerous cases of fibrosarcoma of the larynx are reported in the literature, all of the tumors occurring in adults. Figi [2] in 1933 reported 4 cases of sarcoma of the larynx, in 2 of which the growth was a fibrosarcoma. In 1935 New [3] reported a case of fibrosarcoma of the larynx in a 51 year old man. In a review of the literature on laryngeal tumors of the last thirty years, Havens and Parkhill [4] found 15 cases of sarcoma of the larynx, 10 of which were instances of fibrosarcoma. Their own report included 11 cases of sarcoma, 8 of the tumors beings fibrosarcomas and all occurring in adults. According to Figi [2] and Ewing,[5] the vocal cords and the area around the epiglottis are the usual points of origin of fibrosarcoma, and the tumor is generally pedunculated and lobulated. In the case herein described, the tumor had an extrinsic origin and was neither pedunculated nor lobulated. These factors, together with the age of the patient, are the unusual features emphasized by this report, rather than the not too uncommon type of laryngeal tumor.

700 North Michigan Avenue.

---

2. Figi, F. A.: Sarcoma of the Larynx, Arch. Otolaryng. **18**:21-33 (July) 1933.

3. New, G. B.: Sarcoma of the Larynx, Arch. Otolaryng. **21**:648-652 (June) 1935.

4. Havens, F. Z., and Parkhill, E. M.: Tumors of the Larynx Other than Squamous Cell Epithelioma, Arch. Otolaryng. **34**:1113-1122 (Dec.) 1941.

5. Ewing, J.: Neoplastic Diseases, ed. 4, Philadelphia, W. B. Saunders Company, 1940.

ADENOCARCINOMA INVOLVING THE MIDDLE EAR

EUGEN GRABSCHEID, M.D., NEW YORK

Malignant tumors of the middle ear are rather rare. Among these tumors, adenocarcinoma of the middle ear, either primary or secondary, is one of the most infrequent, as was pointed out by Rosenwasser [1] and by Peele and Hauser.[2] This fact, as well as the further fact that the case to be described differs in many respects from the cases reported in the literature, suggests its publication.

REPORT OF A CASE

E. H., a housewife 47 years old, had noticed poor hearing in both ears at the age of 27. The hearing in the left ear improved after treatment with air inflations and massage, whereas the hearing in the right ear became progressively worse.

In October 1937, the patient observed diminution of hearing and felt intermittent pain in the left ear. The consulted otologist diagnosed acute otitis media and advised conservative treatment. A slight hemorrhagic discharge occurred, which lasted for three days. There was no history of a previous discharge from the ear. The pain continued for one week. As the patient felt a foreign body in the external auditory canal, she came to the dispensary five weeks after the diagnosis of acute otitis had been made.

Examination of the ears revealed the following pertinent data: The right ear drum was dull and retracted. The inner part of the left external auditory canal was filled with a grayish red tissue, firm and uneven. There was no discharge. The ear drum was not visible, and there were no signs of involvement of the mastoid. Accentuated whispered voice was heard at 1.5 meters on both sides. In the Weber test there was no lateralization. The results of the Schwabach test were almost normal on both sides. The Rinne reaction was negative on both sides. Perception of tone from a c-4 tuning fork was shortened on the right side by twelve seconds and on the left by ten seconds. There was no spontaneous nystagmus. The caloric test revealed normal irritability on the right side and hypoirritability on the left side. The turning test did not show any difference in the activity of the two labyrinths. The fistula symptom was absent. There was no past pointing or falling.

Examination of the nose and throat revealed no pathologic changes.

The tissue in the left external auditory canal was removed, but the unusually hard consistency, which was not combined with excessive bleeding, as well as the persistent pain without any signs of an acute exacerbation, suggested the possibility of a tumor. The microscopic examination revealed adenocarcinoma.

From the Ear, Nose and Throat Department of Docent Hans Brunner of the General Polyclinic of Vienna.

1. Rosenwasser, H.: Neoplasms Involving the Middle Ear, Arch. Otolaryng. **32**:38-53 (July) 1940.

2. Peele, J. C., and Hauser, G. H.: Primary Carcinoma of the External Auditory Canal and Middle Ear, Arch. Otolaryng. **34**:254-266 (Aug.) 1941.

The tumor consisted of glandular tubules embedded in dense connective tissue. The lumen of the tubules was partly filled with a fluid stained with eosin. At some places the tubules were surrounded by patches of dense lymphatic infiltration. The tumor was covered by a layer of squamous epithelium which showed only a few projections into the underlying tissue. The glandular tubules almost reached the epithelium (fig.).

The patient was admitted to the hospital, where careful internal, neurologic, gynecologic, ophthalmologic, biochemical and roentgen examination revealed no pathologic conditions. The roentgen examination of the ear revealed pneumatization of both mastoids, with the cells of the right side clear and sharply outlined and those of the left side somewhat cloudy and not clearly outlined. There was no involvement of the regional lymph glands.

Adenocarcinoma of the middle ear (photomicrograph, staining with hematoxylin and eosin).

On Nov. 3, 1937 a radical mastoidectomy was performed. The mastoid showed medium-sized cells. The antrum and middle ear were filled with soft granulations. The horizontal semicircular canal was covered by a firm, thickened membrane which was difficult to remove. After removal of the membrane the bony canal showed numerous spots of petechial hemorrhage. The incus was normal. The handle and the short process of the malleus were destroyed. The tegmen antri and the tegmen tympani were necrotic. The removal of the necrotic tegmen resulted in a broad exposure of the normal dura. The eustachian tube was entirely blocked, so that not even the finest probe could pass. There was no frank pus. The postoperative recovery was uneventful, and the patient was discharged from the hospital thirteen days after the operation.

The microscopic examination of the granulations verified the diagnosis of adenocarcinoma. Massive doses of radiation were given, and the mastoidectomy cavity healed in seven weeks.

The patient was carefully followed up for four years and had no more complaints. The cavity remained healed, and a roentgenogram made three years after the operation did not reveal further destruction of bone beyond the defect caused by the operation.

COMMENT

From the reports of adenocarcinoma of the ear (Fraser,[3] Furstenberg,[4] Gritti,[5] Heschelin,[6] Lange,[7] Lukens,[8] Melan,[9] Peele and Hauser,[2] Rosenwasser,[1] Schall [10]) I omit reviewing the cases in which the origin of the carcinoma was found to be in the external auditory canal extending into the tympanum; further, the cases in which the destruction was so extensive that the origin could not be determined; the cases which were not described in sufficient detail to enable one to determine whether or not the tumor arose primarily in the external canal; and finally, the cases in which the tumor proved to be a secondary growth. Lange [7] described an adenocarcinoma in a patient 83 years of age, who suffered for many years from a discharging ear and attacks of dizziness and facial paralysis. A polyp in the canal was removed with a curet, and the microscopic examination revealed a papilloma. The patient died soon afterward, and the autopsy revealed extensive destruction of the bone of the petrous pyramid with secondary tumors in the regional lymph glands and lungs. The microscopic examination of the granulations in the middle ear and of the secondary tumors in the glands and lungs revealed adenocarcinoma. The carcinoma was characterized by abundant production of mucus. As evidence of the fact that the tumor was a primary one of the middle ear, Lange [7] pointed to the localization, to the nature of the growth and to the extensive destruction which was limited to the middle and inner ears. In spite of these points, it is still questionable whether the tumor masses in the lungs did not represent the primary tumor, since the tumor removed from the ear was characterized by abundant production of mucus and it is known that carcinomas of the lungs show a tendency to form such secondary tumors.

Furstenberg [4] reported the case of a patient 67 years of age who had a purulent, offensive discharge from the left ear. There developed severe pains in the ear, radiating to the tube, pharynx and occiput.

3. Fraser, J. S.: Malignant Disease of the External Acoustic Meatus and Middle Ear, J. Laryng. & Otol. **45**:636-643, 1930.

4. Furstenberg, A. C.: Primary Adenocarcinoma of the Middle Ear and Mastoid, Ann. Otol., Rhin. & Laryng. **33**:677-689, 1924.

5. Gritti, P.: Adenocarcinoma dell'orecchio medio, Oto-rino-laring. ital. **4**: 503-515, 1934.

6. Heschelin, A.: Primärer Krebs des Mittelohres, Acta oto-laryng. **7**:412-418, 1924-1925.

7. Lange, W.: Ein Fall von primärem Zylinderzellenkarzinom des Mittelohres, Ztschr. f. Ohrenh. **46**:209-230, 1904.

8. Lukens, R. M.: Adenocarcinoma of the External Auditory Canal, Ann. Otol., Rhin. & Laryng. **45**:567-573, 1936.

9. Melan, A.: Adenocarcinoma dell'orecchio medio, Valsalva **2**:15-19, 1926.

10. Schall, L. S.: Neoplasms Involving the Middle Ear, Arch. Otolaryng. **22**: 548-553 (Nov.) 1935.

The roentgenogram of the mastoid showed destruction of the cellular structure with the exception of the tip. A radical operation was performed, and the tumor masses found in the middle ear and in the mastoid process were reported to be adenocarcinoma with dense hyaline degeneration of the older parts of the tumor. Careful examination of the patient revealed no other pathologic condition. Roentgen treatment was given, and no recurrence was noticed four and one-half years after the operation. Furstenberg believed the tumor to be caused by a malformation of the mucous membrane following blastomatous transformation. Kolmer [11] reported that he had found widespread columnar epithelium in the tympanal cavities of anthropoids only when they suffered from otitis media, but it is a known fact that acute otitis media in man causes increase in the height of epithelium until it finally reaches a cylindric shape.

The case reported here differs from the more frequent cases of squamous cell carcinoma of the middle ear in the following clinical aspects:

1. There was no history of a chronic discharge from the ear. As a matter of fact, five weeks prior to the patient's admission to the dispensary, a specialist made the diagnosis of an acute otitis, not of a chronic otitis such as is usually found with squamous cell carcinoma. In accordance with that history, the surgical exposure of the middle ear and mastoid process did not reveal frank pus or cholesteatoma.

2. The mastoid processes showed good pneumatization, while with squamous cell carcinoma the pneumatization of the mastoid processes is diminished as a rule.

3. The incus was intact, while the malleus presented destruction of the handle and the short process only. The latter observation suggests that the destructive agent originated in the inferior and anterior part of the middle ear, specifically in the region of the eustachian tube.

The third feature just mentioned points to the conclusion that the tumor originated in the region of the eustachian tube. In favor of that concept is also the following observation: According to Politzer,[12] there are occasionally glands in the anterior part of the tympanal cavity close to the tympanal ostium of the tube, and these glands increase in the direction of the tube. At operation in the present case the tympanal opening of the eustachian tube was found to be entirely blocked by tumor masses, so that it was not possible to pass even a fine probe into it.

Finally, I assume that the adenocarcinoma in this case was a primary one, as careful examinations for four years after the operation failed to reveal a primary focus. This view is supported by the fact that the structure of the tumor indicated a primary growth rather than a secondary one.

I wish, moreover, to point out that the now generally accepted method of treatment, namely operation with subsequent irradiation, proved to be successful.

---

11. Kolmer, W.: Gehörorgan, in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1927, vol. 3, pt. 1.

12. Politzer, A.: Lehrbuch der Ohrenheilkunde, ed. 4, Stuttgart, Ferdinand Enke, 1901.

Clinical Notes; New Instruments and Technics

RECOGNITION AND RADIUM TREATMENT OF INFECTED AND HYPERTROPHIED LYMPHOID TISSUE IN THE NASOPHARYNX

Preliminary Report

GILBERT E. FISHER, M.D., BIRMINGHAM, ALA.

It has been known for years that lymphoid tissue is readily affected by exposure to roentgen rays and radium. Heinlke [1] in 1905, in a study of the biologic effects of roentgen rays on lymphoid tissue, noted that after long exposure of animals to radiation both the circulating lymphocytes and the lymphoid tissue were almost completely destroyed. This observation led to the use of the roentgen ray in the treatment of lymphatic leukemia and in reducing the size of the enlarged spleen and lymph nodes occurring in other diseases.

Akaiwa and Takeshima,[2] in 1930, carried out experiments on the effects of irradiation of the lymph nodes in the popliteal spaces of rabbits. Thirty minutes after exposure to radiation the lymph follicles of these nodes were enlarged. After one hour the nuclei of many of the cells began to show disintegration, which reached its height in from two to six hours. From this time on pronounced phagocytosis was noted, the phagocytes taking up the chromatin particles of the decomposed nuclei. Owing to this disintegration of lymphocytes, there was a considerable decrease in the number of these cells in the lymph follicles after forty-eight hours. Following these phenomena there was a period of regeneration, so that after the seventh day the lymph node resumed its normal appearance except for an increase in the connective tissue. Macroscopically there was an increase in the size of the node after the first two hours following irradiation. There was then a gradual decrease in the size of the node, so that in two weeks the node was smaller than normal.

It would thus appear after these conclusive experiments that if the lymphoid tissue of the nasopharynx could be readily exposed to the rays of radium, this tissue could be thoroughly and painlessly removed from its environment.

In examining hundreds of school children between the ages of 8 and 14 years, Crowe [3] observed a pronounced recurrence of lymphoid tissue in the nasopharynx in more than 75 per cent of those who had previously been subjected to tonsillectomy and adenoidectomy. He expressed the belief that this recurrence was due not to incomplete operation but to the fact that the lymphoid tissue of the nasopharynx and pharynx is an integral part of the mucous membrane and could not possibly be entirely removed by surgical means unless the entire thickness of the mucous membrane were removed. Thus, one is forced to conclude that the recurrence of the lymphoid tissue in the nasopharynx following adenoidectomy, prior to puberty,

1. Heinlke, H.: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **14**:21, 1905.

2. Akaiwa, H., and Takeshima, M.: The Reaction of Lymphoid Tissue to Roentgen Radiation, Am. J. Roentgenol. **24**:42, 1930.

3. Crowe, S. J.: Personal communication to the author.

must be considered a normal reaction. The removal of this large mass of lymphoid tissue prior to puberty also causes compensatory hypertrophy of all the lymphoid follicles in the pharynx, resulting in the "granular pharyngitis" so commonly seen in children. The treatment of this condition does not necessitate a second operation, as the tissue atrophies remarkably when exposed to the gamma rays of radium.

Primary infection of the lymphoid tissue of the nasopharynx may extend to the sinuses, ears, pharynx, larynx and bronchi. This is true in adults as well as in children. The infection leads to a constant irritating postnasal discharge, causing recurrent pharyngitis. Repeated infections result in hypertrophy and hyperplasia of the lymphoid tissue of the nasopharynx, which produce mechanical obstruction of the orifices of the eustachian tubes and in time will lead to chronic tubotympanic catarrh.

In Birmingham, where the atmosphere is frequently filled with a high concentration of smoke and dust, postnasal discharge is an almost universal complaint. Examination of sufferers from this condition with an electric nasopharyngoscope invariably reveals an enlarged mass of hyperemic lymphoid tissue lying in the midline of the nasopharynx and covered with a thick, tenacious discharge. Frequently there will be complete or partial obstruction of the orifices of the eustachian tubes by this tissue. The nasopharynx also presents tremendous hypertrophy of the mucous glands in the mucosa overlying the posterior end of each inferior turbinate.

While I was working under Dr. S. J. Crowe in the department of otolaryngology at the Johns Hopkins Hospital, I saw many persons with this condition of the nasopharynx greatly improved when the nasopharynx was treated with radium. With these facts in mind, I was led to begin a simliar treatment of the patients on whom I am about to report.

INSTRUMENT

The instrument used is a 50 mg. radium applicator. The radium is held in a platinum capsule 0.5 mm. in thickness. This capsule is attached to a copper wire 7 inches (17.5 cm.) long. The platinum capsule and the wire are covered with a coating of gum rubber 0.7 mm. in thickness.

METHOD OF APPLICATION

A 1 per cent solution of cocaine is lightly sprayed into each inferior meatus. A cotton-tipped applicator, moistened with 3 drops of a 10 solution of cocaine, is then gently passed along the floor of each inferior meatus. After an interval of five minutes the radium applicator is inserted through the inferior meatus into the nasopharynx. A 2 gram minute treatment is given to each side. This treatment causes the patient no discomfort whatsoever and can be given to children and adults alike. During the first week following the treatment there may be a slight subjective sensation of "stuffiness" or "fulness" in the nasopharynx, accompanied by a moderate increase in the postnasal discharge. This is followed by a diminution in the amount of postnasal discharge, so that four to five weeks after treatment none is noticeable. Examination of the nasopharynx six weeks after treatment reveals definite diminution in the size of the lymphoid tissue in the nasopharynx as well as a decrease in the amount of tenacious mucoid discharge.

RESULTS

One hundred patients were treated in the manner described and were reexamined six to eight weeks after the treatment. Ninety-four patients stated that their postnasal discharge had ceased. Excessive dryness of the nasopharynx was not complained of in a single instance. Twelve well selected patients with bilat-

eral conduction deafness, caused by long-standing obstruction of the eustachian tubes, noted a decided improvement in their ability to hear as proved by audiometric studies carried out before and after radium treatment. Two patients with severe tinnitus were unimproved except that their postnasal discharge ceased. Four patients who gave a definite history of allergic rhinitis, and whose nasal mucosa presented an edematous appearance characteristic of allergy, were unimproved.

CONCLUSION

1. The recurrence of lymphoid tissue in the nasopharynx following adenoidectomy prior to puberty is so common that it must be considered a normal reaction.

2. Chronically infected and hypertrophied lymphoid tissue in the nasopharynx causes obstruction of the orifices of the eustachian tubes and is constantly covered with a thick, tenacious discharge. Atrophic changes can be readily produced in this tissue by exposing it to the gamma rays of radium.

501-505 Medical Arts Building.

IRRADIATION OF THE NASOPHARYNX IN OFFICE PRACTICE

EDLEY H. JONES, M.D., VICKSBURG, MISS.

Since Crowe[1] and his associates recommended irradiation of the nasopharynx in the treatment of certain types of impairment of hearing, many investigators have substantiated their observations, and some have offered certain modifications of treatment.

The otologic department of the Johns Hopkins clinic used a radon applicator containing 600 millicuries of radon in a special applicator for such treatments.[2] Fowler[3] mentioned having used an applicator containing 250 millicuries of radon. The employment of such large quantities of radon decreases the length of time of application and is therefore more convenient for the patient, but it is obvious that these amounts are available to only a small percentage of the otolaryngologists who would be interested in this treatment. To start with, such large quantities of radium are not universally available; second, there are still fewer radon plants; third, transportation of quantities larger than 100 millicuries of radon is limited to messenger service. Radium cannot be shipped by air express. The Railway Express Company will not accept more than 100 milligrams of radium or 100 millicuries of radon for shipment at one time.

Realizing this situation, Fowler[3] described his technic of using 30 mg. of radium in a brass capsule for irradiation of the nasopharynx. Emerson, Dowdy and

---

1. Crowe, S. J.: Recognition, Treatment and Prevention of Hearing Impairment in Children, Laryngoscope **50**:658 (July) 1940.

2. Polvogt, L. M.: Report of Recent Studies on the Prevention of Deafness, Tr., La.-Miss. Ophth. & Otolaryng. Soc., 1940.

3. Fowler, E. P.: Non-Surgical Treatment for Deafness, Laryngoscope **52**:204 (March) 1942.

Heatly [4] recently described applicators containing 25 mg. of radium. While these procedures are excellent, their employment is obviously limited to hospitals and clinics. So far as I know, no one has previously described a technic that may be used in the office.

In September 1940, at my request, the Radium Emanation Corporation, of New York city, made up an applicator modeled after the one used at the Johns Hopkins

Fig. 1.—Photograph of applicator.

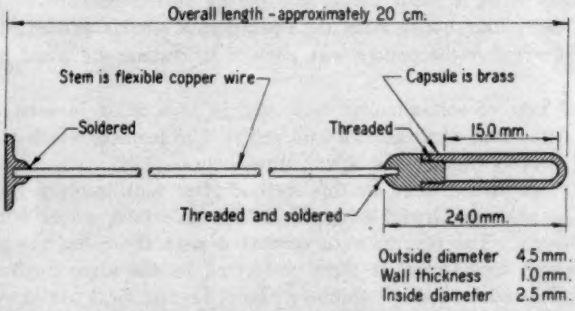


Fig. 2.—Diagram of applicator.

department of otology. This applicator, containing 100 millicuries of radon, can be shipped by express. While the radioactive power of the radon decays with time, this company's service permits the exact use of the applicator. For example, when I schedule a patient for treatment, I can order the applicator and have it delivered

4. Emerson, E. B., Jr.; Dowdy, H., and Heatly, C. A.: Use of Radium in Treatment of Deafness by Irradiation, Arch. Otolaryng. **35**:845 (June) 1942.

at my office at the proper time, accompanied by a certificate specifying the period that it must be applied to deliver the required dose.

I have used this method in my office several times and found it entirely satisfactory. The cost is reasonable.

LOCAL USE OF SULFANILAMIDE IN TONSILLECTOMIES

KARL N. VICTOR, M.D., LOUISVILLE, KY.

Attention was first directed to the local use of the sulfonamide compounds in tonsillectomies after the observation that surgical wounds in which the drugs were employed locally as a therapeutic agent, as in the treatment of osteomyelitis, remained almost free of postoperative oozing. As reported by Cunningham,[1] the sulfonamide drugs used locally after tonsillectomy have a proved hemostatic action.

It was felt that sulfanilamide was the drug of choice in the present experiments, as locally it produces a minimal foreign body reaction with the attendant extravasation of serum and delayed healing of the wound. This drug also persists in the wound for the shortest period of all the sulfonamide compounds, from only six to twenty-four hours, penetrating to a tissue depth of less than 3 mm.,[2] but can be detected chemically for eleven days. Sulfanilamide also is primarily specific for the hemolytic streptococcus, the chief offender in postoperative tonsillar infections, and affects adversely the pathogenic action of various other bacteria.

The method of procedure was as follows: Immediately after the removal of the tonsils a small amount of sulfanilamide powder was held in firm contact by means of a sponge in both the right and the left tonsillar fossa simultaneously for five minutes. Eight hours after the operation a thin layer of sulfanilamide powder was applied directly into the fossa with the aid of a powder blower. The patient was instructed to hold the breath during this procedure so as to avoid coughing. Twenty-four hours after the operation a similar procedure was again carried out. Thereafter the patient was allowed to resume the usual postoperative routine.

Not over 5 Gm. of sulfanilamide was used in each fossa in each of the three procedures. After each application a thin yellow film formed, which was gradually absorbed and entirely disappeared within three hours.

Twenty-five patients treated by this method after tonsillectomy were observed. The operations were performed on children and on adults, under both local and general anesthesia. The results were compared with those for the preceding 25 patients on whom tonsillectomies were performed by the same methods, with the same routine and under similar conditions except for the local use of sulfanilamide. The following observations were made.

1. There was no case of postoperative oozing in the sulfanilamide-treated group, as compared with 2 cases of postoperative oozing in the untreated group.

2. No positive blood cultures were obtained during the first twenty-four hours from the sulfanilamide-treated group, such cultures being taken six and eighteen

---

1. Cunningham, B. P.: Ann. Otol., Rhin. & Laryng. **51**:301-316 (June) 1942.

2. Porter, C. T.: Tr. Am. Acad. Ophth. (1941) **46**:273-278 (July-Aug.) 1942.

hours after operation. From 3 of the 25 untreated patients positive blood cultures were obtained at similar intervals.

3. Leukocytosis was present twenty-four hours after operation in both the treated and the untreated group, but in the former the leukocyte count more nearly approached normal.

4. The average postoperative temperature for the first twenty-four hours for the sulfanilamide-treated group was 99 F. as compared with the average temperature of 99.98 F. for the untreated group.

5. Patients treated with sulfanilamide presented less slough, less inflammatory reaction of the fossa and adjacent structures and less bad taste and odor.

6. There was no case of secondary infection in the treated group, as compared with 1 case of secondary infection in an equal number of the untreated patients.

7. The degree of soreness was lessened in the sulfanilamide-treated patients. Complete convalescence and healing took place several days earlier than in the untreated patients, with considerably less formation of the so-called grayish white healing membrane.

No determinations of the concentration of sulfanilamide in the blood were made, but it was felt that the absorption was slight as no toxic reactions were noted, the action of the drug, probably both bacteriostatic and bactericidal, being entirely local.

COMMENT AND SUMMARY

From the observations stated the local use of sulfanilamide powder in postoperative tonsillectomies apparently offers various advantages.

The small group of patients studied is inadequate for definite conclusions, although such deductions as have been made merit serious consideration concerning the advisability of the routine use of sulfanilamide and should stimulate further study.

It is desired that this report be considered a preliminary one. Further studies related to the subjects are being carried out in the department of otorhinolaryngology at the Louisville General Hospital.

401 Breslin Medical Arts Building.

News and Comment

AMERICAN BOARD OF OTOLARYNGOLOGY

The next examination of the American Board of Otolaryngology will be held in New York, at the Waldorf-Astoria Hotel and the New York Eye and Ear Infirmary, on June 3, 4 and 5, 1943. Candidates should apply at once to Dr. Dean M. Lierle, secretary of the board, University Hospital, Iowa City.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

REVIEW OF ALLERGY FOR 1941

F. L. MacQUIDDY, M.D.
OMAHA

THEORIES

Vaughan and Derbes [1] investigated the relationship between infection and allergy, using 250 patients who were definitely allergic in one way or another and 250 nonallergic patients. They concluded that no significant difference was found in the incidence of acute infectious diseases in allergic and nonallergic persons. This brings up the question of the etiologic and the predisposing factors of "allergy."

Urbach [2] presents various etiologic factors and draws his material from the most important physiologic experiments. 1. Experimental anaphylaxis may be induced at will in animal species with certain antigens. Atopic hypersensitiveness cannot be induced, even by artificial contact, in persons not subject to the atopic hereditary influence (atopy being defined as a type of hypersensitiveness peculiar to man which is subject to hereditary influence). 2. Experimental anaphylaxis follows a previous, intentional exposure to a given antigen. Human "atopy," on the other hand, often seems to occur without any demonstrable previous exposure to incitant substances. 3. The substances which incite human allergies (atopies) are often nonantigenic in the sense that they are incapable of inciting antibody production or of sensitizing animals. 4. Animals can be readily sensitized experimentally. In human beings, on the other hand, it is difficult to achieve intentional sensitization. 5. Experimental anaphylaxis is based on an antigen-antibody mechanism and is characterized by the presence of anaphylactic antibodies. Antibodies are demonstrable by passive transfer of the anaphylaxis to other animals. In allergic human subjects, on the other hand, only antibodies (named reagins by Coca) are demonstrable and can be transferred only from man to man by the Prausnitz-Küstner method.

1. Vaughan, W. T., and Derbes, V. J.: Comparative Study of the Incidence of Acute Infectious Diseases in Allergic and Non-Allergic Persons, J. Allergy **12**:477-480, 1941.

2. Urbach, E.: The Concept of Atopy in the Light of Recent Investigations, Arch. Pediat. **58**:482-494, 1941.

Furthermore, the blood of anaphylactic animals contains precipitins, which are not found in the blood of allergic patients. 6. Human allergy behaves in accordance with the mendelian hereditary formula. Experimental anaphylaxis can be transmitted only passively, and only in the first generation. An antianaphylactic phase, of variable duration, always follows a nonfatal anaphylactic shock in guinea pigs; such a phase is not observed in human beings. 7. Experimental anaphylactic desensitization can be achieved by administration of the exciting agent. In clinical allergy (atopy) such desensitization cannot be achieved. 8. Those forms of human hypersensitiveness which Coca has named atopic are characterized by a specific immediate whealing type of cutaneous reaction, while bacterial hypersensitiveness is characterized by a late reaction of the tuberculin type.

The theory that hypersensitiveness in man and experimental anaphylaxis are only different expressions of a single underlying biologic process dependent on cellular antigen-antibody mechanisms is supported by numerous experimental and clinical observations.

Fineberg [3] reviews the material concerned with the action of therapeutic agents as allergens. Of these, he mentions particularly the reactions to serum, vaccines, drugs and glandular substances. The reactions vary from simple, local edema and irritation to urticaria, rhinitis, asthma, dermatitis, damage to the liver, kidneys and brain, changes in the blood, anaphylactic shock and death. He divides the serum reactions, which are usually caused by horse serum, according to the interval between the injection and the reaction. 1. Serum sickness is an acute condition developing nine to ten days after the injection, being manifested by such signs as dermatitis, urticaria, enlarged nodes, pains in joints and albuminuria. Serum sickness cannot be predetermined, since it occurs as a result of the presence of antigen and the development of an antibody titer. 2. The accelerated reaction comes on in a few hours or days after a second injection of the serum. It is seen in persons who have had serum sickness. 3. The immediate reaction occurs within a few moments or hours and varies from a few sniffles and urticaria to violent asthma, shock and death. This response depends on previous sensitization by serum or its equivalent. A history of allergy or previous injections of horse serum is therefore important.

Of the other organic substances used in injection, insulin may give a local reaction in a few instances. If this occurs a change in brand is necessary. The author reports 2 cases in which sensitization to liver extract developed. This sensitization was probably based on the presence of an organic or a specific protein. Estrogenic substances and vitamins

3. Fineberg, S. M.: Allergy to Therapeutic Substances, Illinois M. J. **80**: 244-249, 1941.

have also been indicated, but in cases of a reaction to such substances the vehicle may have been the cause. Even milk may give a reaction in sensitive persons.

Drugs must also be considered, since those commonly used, such as the antipyretics, morphine derivatives and quinine, are listed as causing asthma, urticaria, abdominal pain, headache and purpura. The barbiturates and the arsenicals likewise occasionally produce a dermatitis. Fineberg also states that asthma due to acetylsalicylic acid is severe and that of 50 patients sensitive to this drug 46 had asthma and the majority had allergic rhinitis, urticaria or angioneurotic edema. In these persons, however, a negative cutaneous reaction to the drug was not uncommon. The toxic symptoms of sensitivity to the sulfonamide compounds are also well known.

Experimental work to determine the relationship of histamine and allergy was done by Rose.[4] He studied the histamine content of the blood of 80 patients with allergic disease, including asthma, urticaria, angioneurotic edema, eczema, vasomotor rhinitis and colitis. He noted the variation in the histamine levels of the blood associated with these various allergic conditions and concluded that although histamine may be released in the types of allergic disease studied in this communication, the results of his observations do not warrant this statement as a fact.

Kaufman [5] made a study of the relationship of histamine, the eosinophil level and allergy, using 22 patients with pollinosis and giving multiple 1 mg. injections to each patient. He studied the eosinophil levels before and after treatment and concluded that the therapeutic value of histamine is not the result of alteration of the relative number of eosinophils.

ALLERGY OF THE EYE

With respect to the particular shock organs of allergy, one may consider the relation of allergy to the eye. Bab [6] has presented a study of allergy from the ophthalmologic standpoint. Of the important allergens, he mentions dusts, smokes, pollens, food, cosmetics, heat, light, dyes, drugs and soaps as well as local antiseptics, astringents (zinc), the base of ointments and eye lotions, particularly a 3 to 10 per cent solution of boric acid. He differentiates hypersensitiveness from mechanical irritation by noting that the former remains longer, the reaction is typically allergic and a repetition of the symptoms is produced with use of the offending substances. These offenders may enter the body by ingestion, injection, inhalation and contact, and the eye may act

4. Rose, B.: Alterations in Blood Histamine in Patients with Allergic Disease, J. Clin. Investigation **20**:419-428, 1941.

5. Kaufman, R. E.: The Effect of Histamine Injections on Blood Eosinophils in Allergic Patients, Yale J. Biol. & Med. **13**:813-816, 1941.

6. Bab, W.: Allergy of the Eye, Am. J. Ophth. **24**:759-767, 1941.

as the shock organ. The allergy may manifest itself in nearly all the anatomic parts of the eye.

The diagnosis depends on close cooperation with an allergy specialist, since tests of exposure and avoidance, cutaneous tests and clinical experience, as well as a good history and clinical examination, are important. Bab states that the main feature of ocular allergy is often subjective hypersensitiveness, including itching, nervousness, insomnia, headache, excess lacrimation and even cardiac palpitation.

He reports observations in 83 cases, in 41 of which there was edema of the eyelids. The etiologic agents varied greatly and included sleeping pills (cyclohexenylethylbarbituric acid [phanodorm]), in 1 case; stewed cherries, in another; strawberries, in 2 others, and mechanical irritation, such as that produced by plucking the eyebrows, squeezing the margin of the lid to straighten the lashes and dyeing the eyebrows.

Bab summarizes the treatment of ocular allergy: (1) Specific or nonspecific desensitization is usually not satisfactory; (2) small doses of calcium may be used orally, but not intravenously, over a long period; (3) the diet must be changed after the allergen is found; (4) epinephrine, given either locally or hypodermically, is of no value; (5) the allergic agent should be avoided, and sleep and rest should be adequate; (6) the eyes should be bathed several times a day with warm saline solution; (7) colored glasses should be worn if necessary.

Further evidence that the eyelids and conjunctiva are shock tissues is presented by Lehrfeld.[7] He considers that vernal (contact) conjunctivitis is a specific form of allergy. He presents the results of a study of 120 cases and calls attention to the chronic conjunctivitis which is frequently the result of allergy to light.

Bowen [8] offers a working classification of conjunctivitis. He believes that the nonallergic possibilities should be considered first and that if these are not adequate allergy may then be the explanation. He divides conjunctivitis into the vernal and the nonvernal type and offers various possible factors, such as bacteria, environment, drugs, cosmetics, foods, pollen and fungi, as the exciting cause. He feels that the treatment of vernal conjunctivitis is expedited by use of both the water-soluble and the fat-soluble antigen and that the practical management of the vernal or the nonvernal type includes not only the office survey but a personal visit to the patient's home and place of employment.

Klüver and associates [9] suggest that ocular manifestations always indicate generalized hypersensitivity. They give a brief review of ocular

7. Lehrfeld, L.: The Eyelids and the Conjunctiva as Shock Tissue in Allergy, Dis. Eye, Ear, Nose & Throat **1**:100-105, 1941.

8. Bowen, R.: Allergic Conjunctivitis, South. M. J. **34**:184-190, 1941.

9. Klüver, H. C.; Coughlan, C. H., and Martin, L. M.: Hypersensitiveness of the Conjunctiva and Cornea, with Report of a Case, J. Iowa M. Soc. **31**:572-576, 1941.

allergy and its treatment and cite Tuft, who indicated that atropine, pilocarpine, butyn and yellow mercuric oxide may act as etiologic factors in producing conjunctivitis in sensitive persons. Cocaine, as well as typhoid vaccine, may also be included in this list.

THE EAR, WITH PARTICULAR REFERENCE TO MÉNIÈRE'S SYNDROME

Atkinson [10] indicates the relation between allergy and otolaryngology with particular reference to Ménière's syndrome. He contends that the cause of the condition may be a vasospasm or a vasomotor disturbance with allergy as the foundation. The evidence for the existence of an allergic group of patients with this syndrome depends mainly on the patients' good response to treatment. Instead of food tests, however, he used histamine cutaneous tests to decide whether or not the condition was of the allergic type. He gives specific criteria to follow. He also reports on 7 patients with Ménière's syndrome who were sensitive to histamine and who were relieved by desensitization.

In another case he produced "histamine attacks" which were similar to Ménière's syndrome about thirty-five minutes after giving the drug. He considers this response to be the result of a reaction in the labyrinth, with local vascular dilatation and increased capillary permeability.

Vascular spasm, the converse of the dilatation previously mentioned, also causes the syndrome. Such a spasm is relieved by nitrites and vasodilator drugs. The physiologic alteration behind the allergic and the vasospastic group is ultimately one of dilatation and subsequent hindrance of the cochlear and vestibular systems, resulting in labyrinthine imbalance. Atkinson offers a differential diagnosis of the allergic and the vasospastic type.

Allergic Type	Vasopastic Type
1. Positive reaction to histamine	1. Negative reaction to histamine
2. Average age 30; ratio of female 8:1	2. Average age 40 +
3. Hearing loss not great; tinnitus not severe	3. More deafness; more tinnitus; vertigo mild to severe
4. Aura some hours before the attacks; symptoms more severe	4. No aura
5. Better hearing after attack	5. Less hearing after attack
Treatment	
1. Removal of offending foods	1. Nicotinic acid
2. Desensitization to histamine	2. Thiamine hydrochloride

Grove,[11] in a short article classifying the various etiologic agents of vertigo, suggests that Ménière's syndrome is a vascular disease. In the treatment of vertigo due to vasomotor instability and increased capillary

10. Atkinson, M.: Observations on Etiology and Treatment of Ménière's Syndrome, J. A. M. A. **116**:1753-1756 (April 19) 1941.

11. Grove, W. E.: Vertigo, Wisconsin M. J. **40**:799-805, 1941.

permeability considerable success has been achieved by the use of a salt-free diet and by the subcutaneous administration of histamine phosphate; symptomatic treatment consisted of rest, bromides, barbiturates and avoidance of specific factors.

Dean Jr.[12] cites authors who feel that edema and water balance are the important factors in Ménière's syndrome; he states that since, according to his observations, the fundamental cause is edema of the labyrinth, a study of the problem of allergy is important.

Horton[13] reports 49 cases of Ménière's syndrome in which treatment was with intravenous injection of histamine, with prompt relief. The dose was 2.75 mg. of histamine diphosphate (1 mg. of histamine base) in 250 cc. of physiologic solution of sodium chloride. This was given by the gravity method at the rate of 50 drops per minute, one and one-half hours being required for the treatment. Injections could be repeated on successive days. To prevent further attacks a maintenance dose equivalent to 0.1 to 0.2 mg. of histamine base was given subcutaneously two to four times a week. Fifteen of the 49 patients were known to be well; this result, therefore, expresses a tendency toward relief. The author feels that ultimately the treatment will be medical, not surgical, but that the time since treatment is not sufficient to justify a decision.

RHINITIS AND SINUSITIS

In a review of 310 cases in which sinal disease was suspected, Orr[14] reports that in 152 instances symptoms of sinusitis were manifested but examination gave negative results. He classifies such a condition as pseudosinusitis and mentions allergy, endocrine imbalances and metabolic disturbances, as well as "overtreatment," as etiologic factors. He feels that roentgenography is most important in the differential diagnosis, but that even the roentgenographic evidence must agree with the clinical picture. He also uses diagnostic irrigation and depends somewhat on the presence of a pale septum to distinguish allergy.

That allergy is important in the practice of the rhinologist is brought out by Hansel,[15] who says his statistics show that in 16 per cent of cases of disease of the ear, nose and throat and in 40 per cent of cases of nasal and sinal disease the basis is allergy. He states that when surgical treatment does not relieve a patient the cause is probably allergic. He

12. Dean, L. W., Jr.: Ménière's Syndrome and Allergy, Internat. Clin. **3**:231-234, 1941.

13. Horton, B. T.: The Use of Histamine in Ménière's Syndrome, Surg., Gynec. & Obst. **72**:417-420, 1941.

14. Orr, E.: Pseudo-Sinusitis, J. Tennessee M. A. **34**:83-87, 1941.

15. Hansel, F. K.: Diagnosis of Allergy of the Nose and Paranasal Sinuses with Particular Attention to Atypical Types, Arch. Otolaryng. **34**:1152-1162 (Dec.) 1941.

differentiates between typical nasal allergy, with its sneezing, itching and pale mucosa, and atypical allergy, which may be easily overlooked and called a common cold.

Shambaugh Jr.[16] depends more on therapeutic than on cutaneous tests; he also lists a few points of difference between infectious and allergic rhinitis. Of 102 consecutive cases of chronic nasal or sinal disease which he reviewed, the condition was essentially nasal with secondary involvement of the sinuses in 51. In 43 of these there was a response to allergic treatment. In the other 51 cases the disturbance was primarily sinal, and in 36 of these a response to treatment for allergy was obtained. He emphasizes that both the infection and the allergic condition must be treated in order to give maximum relief.

Shahan,[17] after a study of 45 patients selected at random from the allergy clinic and thoroughly investigated with regard to twenty points, including age, race, nationality, sex and occupation, makes the following recommendations: (1) removal of offensive inhalants and foods from the patient's environment; (2) employment of immunization or hypersensitization when necessary (six months to a year is required for success); (3) provision of a balanced diet after offensive foods have been eliminated; (4) removal of foci of infection; (5) palliative medical treatment, and (6) change of environment, if possible.

Since there is some vagueness in the diagnostic term "vasomotor rhinitis," Urbach[18] has attempted to make a specific classification on the basis of examination of 579 patients with asthma. He found vasomotor rhinitis in 145 (38 per cent). He offers the term "rhinopathy" to cover all the conditions known as rhinitis and, in turn, differentiates this as allergic rhinopathy, when the cause is specifically allergy, and pathergic rhinopathy, when the cause is a nonspecific hypersensitivity. Allergens to be considered under the allergic type are exogenous substances, i. e., those exerting influence on the nasal mucosa, such as foods or drugs taken by mouth, and endogenous substances, such as products of the endocrine glands, of metabolism or of bacteria in the intestine. For the pathergic type he considers underlying general or nasal infection, local irritation, psychic disturbances and increased hypersensitivity. Treatment of allergic rhinopathy depends on elimination or desensitization, and treatment of pathergic rhinopathy consists in an attempt to lower the hypersensitivity of the nasal mucosa and to increase the patient's resistance. For this the author uses small doses of phenobarbital and breathing exercises (humming while standing, and later while running).

16. Shambaugh, G. E., Jr.: Head Infections in the Practice of Medicine, J. Iowa M. Soc. **31**:373-377, 1941.

17. Shahan, H. I.: Vasomotor Rhinitis: A Clinical Study of Forty-Five Cases, New York State J. Med. **41**:2409-2424, 1941.

18. Urbach, E.: Vasomotor Rhinitis, Arch. Otolaryng. **33**:982-992 (June) 1941.

Sternberg and Sorrell [19] refer to specific occupations in which asthma and vasomotor rhinitis occur when the potentially sensitive subject comes in direct contact with certain antigens, to which he becomes specifically sensitized through this direct exposure. They state that such persons may give positive cutaneous reactions which are not due to chemicals or drugs.

A rather rare occupational form of asthma is presented by Bubert and Manieri,[20] who give a brief review of the literature pertaining to asthma produced by acacia. This substance is a potential sensitizer when there is adequate exposure to it as an inhalant.

The relationship between existing sinusitis and eventual infective asthma is brought out by Grove,[21] who believes that the latter is the result of sensitization to bacteria or to their products in a person with an allergic constitution.

Another working classification of respiratory allergy is given by Clarke Jr.,[22] who divides it into (1) nasal allergy, including seasonal (hay fever) and nonseasonal (catarrhal and vasomotor rhinitis) forms, and (2) bronchial allergy. He believes that nasal allergy may be so mild as to be overlooked by both the physician and the patient, and consequently that the main complaint in an adult may be with reference to the gastrointestinal tract and in children it may be a behavior problem. He thinks that persons can suffer from nose strain as well as from eyestrain.

The methods of detecting sensitivity by applying an antigen directly to the nasal mucosa are briefly reviewed by Vaughan and Derbes.[23] They feel that the cutaneous tests are not always satisfactory for allergic conditions. Briefly, the nasal contact test entails the use of a mirror originally devised by Glatzel to determine the degree of patency of the nasal passages before or after operation and a quantitative determination of the degree of dilatation of the nasal passages. A method of applying allergens to the nasal mucosa by inhalation is also described. The time required for evaporation of moisture from the mirror is considered as a measure of the quantity of air passing through

19. Sternberg, L., and Sorrell, A. H.: Occupational Asthma and Vasomotor Rhinitis, New York State J. Med. **41**:1649-1652, 1941.

20. Bubert, H. M., and Manieri, F. V.: Bronchial Asthma in the Printing Trade: Non Off-Set Solutions (with Acacia), Bull. School Med. Univ., Maryland **26**:25-30, 1941.

21. Grove, R. C.: The Importance of Chronic Sinusitis in the Treatment of Bronchial Asthma, New York State J. Med. **41**:455-458, 1941.

22. Clarke, J. A., Jr.: Respiratory Allergy: Practical Points in Diagnosis and Treatment, J. M. Soc. New Jersey **38**:184-185, 1941.

23. Vaughan, W. T., and Derbes, V. J.: Individual Modifications of the Nasal Contact Test for Allergy, Ann. Otol., Rhin. & Laryng. **50**:1141-1145, 1941.

each nostril; the patency of each side of the nose is noted, and by mathematical calculation the patient's reaction is rated as 1 to 4 plus.

The need for collaboration between the allergist and the rhinologist is again emphasized by Woodward and Swineford,[24] who analyze the records of 128 consecutive private patients with allergic rhinitis. They provide a comprehensive summary, showing the classification, complications, psychologic factors, methods of treatment, degrees of improvement and therapeutic measures responsible for improvement, as well as the probable causes for failure of the treatment. They recommend that rhinologic and allergic methods of diagnosis and treatment be used and suggest that after the clinical diagnosis has been made the patient may be classified as having (*a*) no complications, (*b*) simple mucous polyps, (*c*) acute or subacute sinusitis, and so on. They emphasize that surgical treatment does not cure vasomotor rhinitis.

The relationship between asthma and foci of infection in the upper respiratory tract is noted by Turnley,[25] who feels that the prophylaxis of asthma includes removal of adenoids, tonsils and nasal obstruction, in addition to careful treatment of the sinuses. He sent out questionnaires to a number of physicians, and his interesting figures are recorded.

Another attempt to clarify the relationship of otolaryngologic disorders and allergy by a review of case records is made by Woodward.[26] He estimates that the frequency of allergic conditions in patients with otolaryngologic disturbances is between 20 and 40 per cent. Fifty per cent of the patients referred to the department of otolaryngology had vasomotor rhinitis only. The condition of most of these persons was controlled by treatment for allergy, and only local symptomatic therapy was used. Fifteen per cent needed a minor operation, such as removal of polyps, and 35 per cent required a more radical operative procedure.

Smith[27] divides his patients into those with an early, preasthmatic condition, who complained of paroxysmal sneezing, blocked nose and rhinorrhea, and those with established bronchial asthma and associated pathologic changes in the respiratory tract. He believes that if an allergic reaction does exist, early removal of the irritant will allow the membrane to return to normal. Continued irritation will result in permanent pathologic changes, and consequently local treatment of

---

24. Woodward, F. D., and Swineford, O., Jr.: Allergic Rhinitis: Analysis of One Hundred and Twenty-Eight Cases in Which the Rhinologist and Allergist Collaborated, Arch. Otolaryng. **34**:1123-1132 (Dec.) 1941.

25. Turnley, W. H.: Bronchial Asthma in Relation to Foci of Infection of the Upper Respiratory Tract, Arch. Otolaryng. **33**:767-775 (May) 1941.

26. Woodward, F. D.: Relationship of Otolaryngology to Allergy, Tr. Am. Laryng., Rhin. & Otol. Soc. **46**:364-369, 1941.

27. Smith, A. T.: Management of Nose and Paranasal Sinuses in Asthma, Pennsylvania M. J. **45**:41-43, 1941.

allergic respiratory disease is futile so far as permanent cure is concerned. The Kings[28] also feel that the condition of about one third of the patients who consult the otolaryngologist is allergic in origin.

Local therapy is suggested by Thacker and Hauser,[29] who claim that 58 patients with chronic simple, hypertrophic vasomotor rhinitis were relieved by submucosal injections of a 5 per cent solution of sodium morrhuate. Eight months has elapsed, and there are no recurrences.

Sternstein[30] gives the result of experimental work with 42 patients and draws the following conclusions: 1. Air pressure of about 15 pounds (6.8 Kg.) gives little or no response of erectile tissue in a non-obstructed nose. The same pressure in an obstructed nose increases the swelling of the erectile tissue and the obstruction. 2. A 0.5 to 5 per cent solution of sodium chloride produces no response in the non-obstructed nose. In the obstructed nose isotonic and hypertonic solutions gradually give nasal obstruction. A 5 per cent hypertonic solution, however, decreases swelling of the turbinates and reduces nasal obstruction.

A summary of the causation and treatment of vasomotor rhinitis is presented by Stovin.[31] His general treatment consists of psychotherapy, drug therapy and glandular therapy as indicated, and local treatment includes zinc ionization, phenolization, electrocoagulation and irradiation.

THE ALLERGIC GASTROINTESTINAL TRACT AND THE EYE, EAR, NOSE AND THROAT

The problem of the all-important "common cold" again comes to the fore as Coca[32] proposes the idea that food allergy is one of the nonspecific factors predisposing to susceptibility to "colds." He cites the work of Locke, who suggests that individual physiologic differences in rabbits partially account for their susceptibility to disease. Locke showed that normal rabbits differed in the speed with which injected type 1 pneumococci were removed from the blood stream. The rabbits which removed the bacteria slowly did not survive. He also showed that animals with a slow "warming time" (time required for recovery of

28. King, E., and King, J.: Diagnosis of Chronic Sinusitis and Allergic Rhinitis, Ohio State M. J. **37**:433-436, 1941.

29. Thacker, E. A., and Hauser, G. H.: Sodium Morrhuate Therapy for Chronic Simple, Vasomotor and Hypertrophic Forms of Rhinitis, Arch. Otolaryng. **33**:600-617 (April) 1941.

30. Sternstein, H. J.: Quantitative Effects of Physical and Chemical Agents on the Erectile Tissue Response in the Nose, Arch. Otolaryng. **34**:523-532 (Sept.) 1941.

31. Stovin, J. S.: Vasomotor Rhinitis: Causation and Treatment, Arch. Otolaryng. **34**:736-742 (Oct.) 1941.

32. Coca, A. F.: The Common Cold: Allergy as a Predisposing Factor, J. Lab. & Clin. Med. **26**:757-765, 1941.

the body temperature from 96 to 100 F.) were unable to remove bacteria from the blood quickly enough to survive. Coca also mentions Locke's work on oxygenation and ventilation and their correlation with the "common cold." The slow use of oxygen tended to result in more "colds," and it was found that oxygenation was impaired at the onset of a "cold." Coca, from his own work with 52 "cold-free" patients and 51 patients with one cold a year, concludes that 90 per cent of persons susceptible to "colds" are allergic. The mechanism is not explained.

Gatewood[33] also considers the relationship between the gastrointestinal tract and allergy of the eye, ear, nose and throat and suggests that when organic causes are eliminated and treatment for a rhinologic condition is not satisfactory, a gastrointestinal disturbance must be considered as the etiologic factor. Some of the more common complaints for which no pathologic basis can be found in the ear, nose and throat are: (1) persistent postnasal drip without sinus disease; (2) vertigo and fulness in the ears without a pathologic condition in the ears to account for such symptoms; (3) rhinorrhea with sneezing, recurring colds, cough and other manifestations of hay fever; (4) dryness and stuffiness of the nasal mucosa with headache, "burning eyes" and a sensation of pressure at the root of the nose; (5) ocular palsy involving the extraocular muscles, with frontal headache; (6) the "lower half headache" of Sluder, consisting of pain about the eye, the upper jaw and the teeth, extending to the zygoma and temple, with earache and pain in the mastoid, extending thence to the occiput, neck, shoulder, scapula, arm, forearm, hand and fingers. He contends that these complaints are on the basis of intestinal toxemia, as shown by the presence of indican in the urine. He believes that incompletely digested foreign protein in the circulation is the fundamental cause and that the difference between a normal and an allergic person is one of degree only. He reports on 4 patients with the symptoms already mentioned who were treated by means of diet and administration of sodium ricinoleate and were advised to eat slowly. All were relieved.

Informative experimental work was done by Walzer,[34] who used rhesus monkeys, human subjects, reagins from clinically sensitive patients and food allergens for his study. From this work he concludes that it is unlikely that the epithelial cells of the various mucous membranes of the body are the seat of allergic reactions. Typical application of an allergen to the surface of the sensitized rectal or intestinal mucous membrane did not elicit a maximal allergic reaction, an experimental observation similar to that noted by Sherman and associates in 1937 in

33. Gatewood, W. L.: Symptoms of Gastrointestinal Origin in Ear, Nose and Throat, Arch. Otolaryng. **33**:592-599 (April) 1941.

34. Walzer, M.: Allergy of Abdominal Organs, J. Lab. & Clin. Med. **26**: 1867-1877, 1941.

connection with the passively sensitized nasal mucous membrane. The fact that the blood-borne antigen, even when diluted, was able to elicit a more intense allergic response than concentrated antigen applied directly to the epithelial surface tends to minimize the importance of the latter as a shock tissue.

MOLDS AND ALLERGY OF THE RESPIRATORY TRACT

That molds continue to be of interest in respiratory allergy is shown by the number of articles written about them.

Harris,[35] using controlled mold room experiments (Alternaria), concludes that a typical history of exposure to molds, in the presence of a positive reaction to the scratch test, probably indicates clinical sensitivity to molds.

Experimental work on the species specificity of Alternaria was done by Pratt,[36] who employed direct testing of patients with different species of Alternaria and also in vivo and in vitro neutralization experiments with the serums of these patients. He concludes that thorough testing of children and adults with several species of Alternaria, varying in culture characteristics and biologic sources, indicates a common atopic excitant in the genus Alternaria. He also suggests that the genus Alternaria is relatively homogeneous in relation to human atopic immunology.

An air survey and clinical study were made by Waldbott, Blair and Ackley[37] in an attempt to determine the relation between fungus spores and allergic diseases of the respiratory tract. They used 841 patients with this type of allergy. They found that cutaneous reactions to one or more fungi were positive in 69 per cent of these patients and that the strongest reactions to fungi occurred in patients who also reacted to other antigens. Cultures from nasal and bronchial secretions showed that Penicillium, Alternaria, Monilia and yeasts prevailed. October and November were the months in which fungi grew most abundantly from the respiratory secretions. They decided that respiratory allergy was rarely due to a single fungus, but that fungi are generally complicating factors in multiple sensitivity associated with asthma and vasomotor rhinitis. In some of their patients the condition was found to simulate merely asthma and vasomotor rhinitis and represented a true fungous infection.

---

35. Harris, L. H.: Specific Reproduction of Respiratory Mold Allergy, J. Allergy **12**:279-289, 1941.

36. Pratt, H. N.: Species Specificity of Alternaria in Asthma and Hay Fever, J. Allergy **12**:431-437, 1941.

37. Waldbott, G. L.; Blair, K. E., and Ackley, A. B.: An Evaluation of the Importance of Fungi in Respiratory Allergy, J. Lab. & Clin. Med. **26**:1593-1599, 1941.

ACCIDENTS IN ALLERGY

An interesting article in *Lancet* [38] refers to unexplained anaphylactic reactions following transfusion. It is contended that hypersensitivity can be transferred by serum injection and that the first transfusion produces the sensitivity. The second transfusion from the same donor then causes the reaction. It is important, therefore, to question all donors concerning a history of allergy, especially if their blood is to be used for the same patient more than once. The use of blood banks, naturally, reduces the possibility of this accident.

An interesting case is reported by Francis,[39] that of a pregnant woman who was treated with grass pollen. There was no grass pollen in the air; yet severe hay fever followed injection of the substance. She had cramps and pain in the lower part of the abdomen with vaginal bleeding, and this was followed by an abortion three days later. He believes that this case proves that systemic reactions can be produced if the dose of the allergen is great enough.

In concluding, the use of the sulfonamide drugs in treatment of allergy may be mentioned. Unger [40] reports on 23 adults with severe, intractable asthma who were treated with one of the sulfonamide compounds. Sixteen of these patients were in the hospital. Only 4 of the 23 patients were improved, and 19 had no relief. Consequently, he concludes that these drugs are to be used for allergic conditions only on the assumption of the existence of secondary infection.

478 Aquila Court.

38. The Allergic Donor, editorial, Lancet **2**:163 (Aug. 9) 1941.

39. Francis, N.: Abortion After Grass Pollen Injection, J. Allergy **12**:559-563, 1941.

40. Unger, L.: Sulfonamide Drugs in Bronchial Asthma: Preliminary Report of Twenty-Three Cases, J. Allergy **12**:528-536, 1941.

Abstracts from Current Literature

Pharynx

PARACOCCIDIOIDES GRANULOMA OF THE BUCCAL, RHINOPHARYNGEAL AND LARYNGEAL REGIONS. A. CELIS PÉREZ, Rev. de la Policlinica, 1941.

Pérez presents 3 cases of granulomatous lesions about the pharynx and larynx due to infection with Paracoccidioides. Lesions due to this infection have been classified into (*a*) the tegmental and (*b*) the lymphatic-ganglionic types. His cases all belong to the first type. The lesions involved the roof and sides of the mouth, the soft palate and the pharynx, the dorsum of the tongue, the hypopharynx and the larynx.

Pathologically, the individual lesion appears as a small ulcer with sharply defined elevated borders. It is usually full of pus and bleeds easily to the touch. After the abscess ruptures, the underlying tissue is hard and indurated. Both the pathologic process and the symptoms are somewhat influenced by the localization of the lesion. Lesions in the mouth and on the palate interfere with the opening of the mouth, while those on the dorsum of the tongue interfere both in speech and in deglutition.

The differential diagnosis between this infection and syphilis, leishmaniasis, cancer, leukemia and tuberculosis must be established. The last three conditions are usually easily eliminated, but it is somewhat more difficult to rule out a diagnosis of syphilis, since a serologic test is frequently positive in cases of the paracoccidioidal infection. Should an intensive antisyphilitic regimen fail to influence the progress of the lesions, a diagnosis of infection with paracoccidioides can be established. Furthermore, a biopsy will help in the differential diagnosis.

In conclusion, the author believes that this infection is endemic in Venezuela. While in 1 of his cases pulmonary lesions were present, he does not believe that they are primary. The clinical aspects of the lesion are generally sufficient for a tentative diagnosis, and this can be made conclusive by biopsy. Finally, he advocates intensive and prolonged use of sodium iodide as perhaps the only drug which, in his experience, has produced any effect on the lesions.

PERSKY, Philadelphia.

Larynx

DIAGNOSIS OF CARCINOMA OF THE ESOPHAGUS. JAMES B. COSTON and WILLIAM T. K. BRYAN, Illinois M. J. **80**:274 (Oct.) 1941.

One reason for the extremely bad prognosis in cases of carcinoma of the esophagus is the late diagnosis of the disease. Improvement can be hoped for only by more intensive search for the tumor in its early stages. Early diagnosis can be obtained only at the expense of routine complete examinations in all cases of unexplained dysphagia, pain in the chest, hoarseness or vomiting. It is unfortunate that the primary symptom, difficulty in swallowing, is common to other esophageal diseases. Hoarseness is to be considered since it might be due to compression from an esophageal growth. The differential diagnosis should include simple hyperacidity of the stomach, cardiac lesions, esophagitis, Plummer-Vinson disease, globus hystericus and diverticulum of the esophagus. The symptoms may be combined later with loss in weight.

In a series of 106 patients, only 4 were examined during the first month of appearance of symptoms. There was marked preponderance of males—92: 14. The average survival of 48 patients whose date of death was known was seven

and six-tenths months; this illustrates the short duration of the disease in the majority of cases and points to the urgent need for prompt action by the patient and the physician.

LEWY, Chicago.

Nose

SHORT WAVE THERAPY IN THE TREATMENT OF SINUSITIS AND ALLERGIC RHINITIS. G. R. BRIGHTON, W. B. SNOW and H. S. FRIEDMAN, J. A. M. A. **118**:507 (Feb. 14) 1942.

The authors conducted a statistical survey of 25 cases of allergic rhinitis, 25 cases of acute purulent sinusitis, 38 cases of chronic purulent sinusitis and 72 cases of chronic nonpurulent sinusitis. They summarize their observations as follows: 1. Short wave therapy is employed indiscriminately for many intranasal conditions in which it does little good, and may do harm. 2. It is of no value in cases of allergic rhinitis. 3. Its application in cases of acute sinusitis, even in conjunction with nasal shrinkage and irrigations, does not produce results superior to conservative nasal therapy alone. 4. In cases of chronic nonpurulent sinusitis, which present in general a poor prognosis, it is of benefit, particularly ih the relief of headache.

GORDON, Philadelphia.

VASOMOTOR RHINITIS: A CLINICAL STUDY OF 45 CASES. HENRY I. SHAHON, New York State J. Med. **41**:2419 (Dec. 15) 1941.

Forty-five patients with run-of-the-mill cases of vasomotor rhinitis were taken from the allergy clinic of the New York Post-Graduate Medical School and Hospital. Adults all, they were studied with twenty points in mind, namely, to remove all causative factors from the alimentary and respiratory tracts; to immunize or hyposensitize for from six months to one year; to eliminate all foods giving a positive cutaneous reaction, but to avoid dangerous elimination through starvation; to remove all focal infections; to administer medicines, useful drugs, vitamins, glandular extracts, etc.; to change the climate when indicated; to manage exposure to such factors as dampness, pillows, pets, face powders, dust, perfumes, automobile rides and crowds, and, finally, not to lose sight of the constitutional factors while correcting local disorders, such as sinuitis—in short, to do away with all health-impairing influences and to "restore these poor unfortunates to as nearly normal a state as possible." Eleven references are cited in support of Shahon's thesis.

VOORHEES, New York.

OSTEOMYELITIS OF THE MAXILLA FOLLOWING SINUSITIS: REPORT OF A CASE. P. N. PASTORE and H. I. LILLIE, Proc. Staff Meet., Mayo Clin. **16**:61 (Nov. 26) 1941.

The most common causes of osteomyelitis are infection and trauma. Both of these conditions are present after an infected antrum has been treated surgically. Only rarely, however, does osteomyelitis of the maxilla follow. In the case reported this complication occurred on the fifth day after a Caldwell-Luc operation. Management consisted of the application of moist warm compresses, sedation and administration of (2-[paraaminobenzenesulfonamido]-pyridine) sulfapyridine. Incision and drainage were carried out as abscess formation was noted.

Osteomyelitis of the maxilla differs from that occurring in the frontal bone, as it usually tends to localize and evacuate itself, because of the thinness of the bone.

The disease was followed by periodic roentgen and clinical examination, and sequestrum formation was found present in about three months. Sequestrectomy at that time was followed by cure. The authors believe that in the prophylaxis of osteomyelitis of the maxilla following operations for sinusitis the following are of major importance: avoidance of trauma to the nasolacrimal duct, insurance

of minimal trauma to the periosteum and preservation of the blood supply. For treatment, early drainage of soft tissue abscesses and a policy of watchful waiting for sequestration give the best results. Chemotherapy is of definite aid in combating potential and actual complications. H. A. MILLER, Philadelphia.

OZENA. ARTURO TELLO, Rev. otorrinolaring. **1**:43 (Dec.) 1941.

Tello has made a comprehensive study of the effect of parenteral injections of double-distilled water on a number of conditions that seem to him to be more or less allied. Among these he groups true ozena, the ozena in tuberculosis, the essential neuralgias, asthma and rheumatism. He devolved this method of treatment from originally using a number of preparations in an attempt to modify various subjective symptoms, particularly pain, in the conditions just named. He employed such preparations as tuberculin, double-distilled water, procaine hydrochloride and histaminase. Observing the action of these, he was struck by the definite amelioration of the symptoms of ozena.

Ozena, he believes, is closely related either by heredity or by direct association with rheumatism, asthma, neuralgia, gout and arthritis; it often assumes the picture of an allergic diathesis.

The use of double-distilled water, he has found, leads to disappearance of frontal pain, fetid odor and crusting. However, in the majority of his cases the improvement lasted only a relatively short period after the termination of the treatment.

Among local changes, he stresses (1) the hypertrophy of the mucous membrane with hypersecretion; (2) the atrophy of the mucosa and of the skeleton; (3) the formation of crusts. He believes that the condition is essentially a reaction to a nonspecific allergin, which may vary, depending on a disturbance of the endocrine, the neurologic or the vegetative system, and that, furthermore, it may be a local anaphylactic phenomenon similar to the Arthus phenomenon.

As a result of the sudden interest in the use of tuberculin in the treatment of ozena, a special commission was named by the Chilean Society of Otorhinolaryngology to investigate this particular problem. This commission had four questions in view: 1. Is there any specific action of tuberculin on ozena? 2. Is there any action on the odor, the crusting and the state of the mucous membrane? 3. Is this action transitory or permanent? 4. What action does tuberculin administered for ozena have on the general state of the patient? As a result of this investigation, they have concluded that a number of patients were wholly cured; that a greater number were definitely improved; that the foul odor and the headaches were among the easiest symptoms to cure; that the crusts themselves seldom disappeared but that, owing to an increase in the secretion in the nose, they became easier to remove and recurred less frequently; that the duration of recovery is uncertain and ever changing but for the time being can be said to be short. However, further studies are necessary to confirm this opinion. Lastly, the general state of the patient is invariably improved. PERSKY, Philadelphia.

Miscellaneous

EMOTIONAL FACTOR IN BRONCHIAL ASTHMA IN CHILDREN. REYNOLD A. JENSEN and ALBERT V. STOESSER, Am. J. Dis. Child. **62**:80 (July) 1941.

Jensen and Stoesser report the cases of 4 patients between the ages of 9 and 16 years whose chief complaint was asthma. All of the 4 children were girls. All were described as highly nervous, under definite emotional tension and anxious. The authors define asthma as a "disturbance of respiration in which there is bronchiospasm and excessive secretion of mucus." They believe that psychic factors in and of themselves cannot cause asthma but that they may be significant in the disease. They emphasize the importance of recognizing and dealing with

the emotional factors in all cases of asthma, but particularly those in which the disease is refractory to the usual types of therapy. They stress the fact that psychologic factors should not be dealt with as a last resort, but that one should take cognizance of their importance and deal with them early in the treatment of asthma, while treatment of the physical manifestations is in process.

WAGGONER, Ann Arbor, Mich. [ARCH. NEUROL. & PSYCHIAT.]

ROENTGENOSCOPY OF PHARYNX IN MYASTHENIA GRAVIS BEFORE AND AFTER PROSTIGMINE INJECTION. R. S. SCHWAB and H. R. VIETS, Am. J. Roentgenol. **45**:357 (March) 1941.

Since dysphagia occurs in about 60 per cent of patients with myasthenia gravis and since it is the third most common symptom in the disease, it seemed important to Schwab and Viets to recognize dysphagia when it first occurs and to estimate how severely the pharyngeal muscles are involved. The authors took advantage of the fact that in myasthenia gravis the pharyngeal muscles are affected by prostigmine to demonstrate the response to the drug in 19 patients by roentgen visualization of the pharyngeal cavity during ingestion of barium sulfate. A group of patients with other forms of dysphagia were similarly studied. Of the 19 patients so examined, 17 responded so strikingly to an injection of prostigmine methylsulfate that practically no barium sulfate was retained in the pharynx. The same patients had also done well under treatment with prostigmine bromide by mouth. The technic of roentgenoscopic examination is described. In a case of severe form, before the intramuscular injection of prostigmine methylsulfate, the thin barium sulfate will be retained in the pharynx in such a manner as to be obvious on the roentgenogram. Fifteen minutes after injection, it is only in patients with myasthenia gravis that the barium sulfate passes through the pharynx in a normal manner. The degree of improvement in swallowing, as demonstrated by this method, is an excellent adjunct to the "prostigmine test." J. A. M. A.

HEADACHES OF OTORHINOLOGIC ORIGIN AND CERTAIN REFLEXES CAUSED BY FIFTH NERVE IRRITATION. JOHN G. MCLAURIN, Ann. Otol., Rhin. & Laryng. **50**:469 (June) 1941.

McLaurin briefly reviews the anatomy of the nose, sinuses and ear, with particular reference to the sensory nerve supply to these parts, before considering headaches of otorhinologic origin and reflexes caused by irritation of the fifth nerve. In cases of headache of nasal origin abnormal conditions in the nasal and paranasal spaces and disturbances of the adjacent nerve structures must be considered. In cases of sinusitis the absence of pain proves nothing, but its presence is important. The degree of pain is not an index to the seriousness of the sinal involvement. Headache may come from pressure due to such marked swelling within the sinus that the membrane of one wall comes in contact with the opposite wall. Pressure may also result from the ostium of a sinus becoming partially or completely closed, causing stagnation of inflammatory products within the cavity; thus the pressure of pus would be exerted against a swollen, sensitive mucosa. Negative pressure may cause headache, and this may occur if the ostium of a sinus is swollen shut. Whether or not a vacuum is produced by closure of the ostium, there would certainly be marked swelling of the lining mucosa, and probably some fluid transudate thrown into the sinus would cause enough pull, and possibly sufficient direct pressure as well, on the sensory nerve endings to produce pain. The occurrence of pain in many sinuses appears to be attributed to involvement of sensory nerve endings in the diseased membranes and not to pus under pressure. The author then discusses headaches in relation to the various sinuses. Acute sinal disease is nearly always associated with pain, but chronic sinusitis may exist without any history of headache. He also discusses headache arising from intranasal pressure, Sluder's "lower half headache," the superior

cervical sympathetic syndrome, suboccipital pain and headaches from malocclusion or defects of the mandibular joint. In addition, he describes a new syndrome accompanying anterior ethmoiditis and pressure on the anterior half of the middle turbinate and causing pain in the upper three or five front teeth, which is relieved by cocainizing the anterior half of the middle turbinate and the middle meatus.

M. V. MILLER, Philadelphia.

DEVELOPMENT OF ROTARY-VESTIBULAR REACTIONS OF THE HUMAN INFANT. MYRTLE B. MCGRAW, Child Development **12**:17 (March) 1941.

A study was made on 67 infants, from birth to 2 years of age, of changing vestibular and ocular adjustments to bodily rotation. Characteristic changes in these adjustments were classified into three phases of developmental significance:

1. Lateral deviation phase: In the newborn infant both head and eyes tend to deviate in the direction of rotation and to remain in the deviated position.

2. Gross oscillatory phase: In the infant a few months old the face generally remains forward, but the eyes make gross, slow horizontal oscillations.

3. Fine oscillatory phase: The older infant shows either fine rapid oscillations during rotation or no detectable response during rotation but equilibratory disturbance subsequently when placed on his feet. The shift from phase 1 to phase 2 in the greatest percentage of children occurs about the end of the fourth month and corresponds to the beginning of object vision. The onset of phase 3 occurs around the seventh month and corresponds to the beginning of equilibratory control in such activities as maintenance of a sitting posture. It becomes established about the end of the second year, which is also the time at which the child manifests optimum equilibratory control. It is considered doubtful that the cerebral cortex exercises much influence on these labyrinthine reflexes, and the development observed probably reflects maturation of the subcortical nuclei.

DAMMANN, Washington. [AM. J. DIS. CHILD.]

SULFANILAMIDE IN THE MANAGEMENT OF ACUTE STREPTOCOCCAL, PARTICULARLY SCARLATINAL, INFECTIONS OF THE UPPER RESPIRATORY TRACT. CONRAD WESSELHOEFT, New England J. Med. **224**:221 (Feb. 6) 1941.

Scarlet fever offers an excellent opportunity to investigate the action of sulfanilamide. In eight clinical studies, comprising a total of 1,239 patients tested against 1,220 controls, this drug was given as a routine by mouth throughout the febrile stage in the accepted dosage. It failed to benefit the treated ones in any way in the eruptive stage. However, if scarlet fever antitoxin or convalescent serum was administered within the first twenty-four hours, one usually obtained a dramatic drop in the fever, a blanching of the rash and a distinct improvement in the inflamed throat.

The next question is whether sulfanilamide can prevent pyogenic complications. In four of these clinical tests administration of the drug was continued only during the febrile stage. The combined results of the tests (474 treated patients; 587 controls) showed no diminution in the incidence of complications. In England, a combination of chemotherapy with administration of scarlet fever antitoxin was used in 317 cases and controlled by 329 cases in similar age groups in which antitoxin alone was given. Again no diminution of the complications occurred as a result of the administration of sulfanilamide.

In contagious diseases not of streptococcic origin the hemolytic streptococci are, nevertheless, frequently the cause of complications. Thompson and Greenfield used sulfanilamide in the treatment of measles and of whooping cough in the usual dosage, followed by a maintenance dose for carefully controlled age groups. The drug showed no effect whatever on the eruptive phase of measles or on the

paroxysmal manifestations of pertussis, but the incidence of bronchopneumonia was reduced in both groups in the drug-treated series. There was a material reduction in the incidence of otitis media in the 400 patients with measles who were given sulfanilamide.

Sulfanilamide is exceedingly valuable in reducing the mortality that follows the onset of serious complications. The majority of the deaths from scarlet fever today are due to the end results of pyogenic infections, particularly of the mastoid cells, in which the blood stream or the meninges become invaded. The death rate for these two serious complications, bacteremia and meningitis, has been markedly reduced.

GENGENBACH, Denver. [AM. J. DIS. CHILD.]

THE 1937-1938 EPIDEMIC OF ACUTE ANTERIOR POLIOMYELITIS IN NEW SOUTH WALES, WITH SPECIAL REFERENCE TO CHANGE IN AGE INCIDENCE, POLIOMYELITIS DURING PREGNANCY AND POLIOMYELITIS FOLLOWING TONSILLECTOMY. KAREN HELMS and W. J. WILLCOCKS, M. J. Australia **1**:467 (April 19) 1941.

Helms and Willcocks discuss certain epidemiologic features of the 1937-1938 epidemic of acute anterior poliomyelitis in New South Wales. During the first nine months of 1937 the average monthly incidence was 2 cases. Between October 3 and October 30 5 cases occurred in the metropolitan area and 7 in rural districts. Up to Sept. 30, 1938 there was a total of 711 cases. The epidemic reached its peak earlier in the metropolitan area than in the rest of New South Wales. A study of the meteorologic elements from October 1937 to September 1938 suggests that the temperature and absolute humidity curves exhibit the closest correlation with the increase and decrease in the number of cases. Humidity, commencing in the spring, attains a maximum in a midsummer month and then declines. A somewhat similar correlation was suggested in the incidence of seasonal rainfall in northern parts of New South Wales. In the metropolitan area, during the early weeks of the epidemic and while the weekly incidence was increasing, severe paralysis developed in a greater proportion of patients than during the later stages of the epidemic. In rural districts the tendency may be masked by the fact that a relatively small population is spread over a large area. The number of deaths was greater in the rural districts at the beginning and at the height of the epidemic than in the later stages. In the metropolitan area there appears to be a tendency for the age group up to 4 years to be proportionately larger and the group more than 15 years of age to be proportionately smaller at the beginning of the epidemic than in its later stages. This tendency may be masked in rural districts for the same reason that the severity of paralysis was masked. The fatality rate in New South Wales was 2.5 per cent, or 17 deaths. For the rural districts it was 4.08 per cent, or 11 deaths. The oldest patient was 63 years and the youngest 7 months of age; 64 per cent of 601 patients were males and 34 per cent females. There were only 5 patients less than 12 months of age. The percentage of patients less than 10 years of age was greater in the metropolitan area (paralytic patients 76.8 per cent) than in rural districts (paralytic patients 53.5 per cent). In the 1937-1938 epidemic of poliomyelitis in New South Wales 5 patients contracted the disease during pregnancy—1 in the fifth, 2 in the sixth, 1 in the eighth and 1 in the ninth month of gestation. A study of these cases and of 13 others reported in the literature showed only 1 in which spontaneous abortion followed closely on the infection. In 13 of the 15 cases in which pregnancy continued beyond the seventh month, the delivery and the child were normal. In the other 2 cases cesarean section was performed because of respiratory paralysis. Poliomyelitis in pregnancy uncomplicated by respiratory paralysis is not an indication for interruption of the pregnancy. In the presence of respiratory paralysis during the last weeks of pregnancy, respiration

may be facilitated by the removal of the fetus. Of the 711 patients, 7 were known to have undergone tonsillectomy and adenoidectomy from seven to twenty-two days prior to the onset of the illness. Bulbar and spinal paralysis developed in 3 of these 7 patients, 2 had paralysis of the palate, 1 had spinal paralysis with no evidence of bulbar involvement and 1 had the nonparalytic form of the disease. There were no deaths. A review of the literature supports the contention that operations on the throat and nose favor the development of poliomyelitis of the bulbar type. When this disease is prevalent, the risk of subsequent invasion by the virus should be well weighed before tonsillectomy or adenoidectomy is undertaken.

J. A. M. A.

SULFAPYRIDINE AS A PROPHYLACTIC AGAINST CEREBROSPINAL MENINGITIS. F. G. GRAY and J. GEAR, South African M. J. **15**:139 (April 12) 1941.

According to Gray and Gear, a bacteriologic investigation of an outbreak of cerebrospinal meningitis in a military camp in Natal, South Africa, revealed approximately 22 per cent carriers among those examined, making conditions favorable for a widespread epidemic. As sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) is known to sterilize the cerebrospinal fluid of patients suffering from meningococcic meningitis within three days, it was thought that it might be equally effective in destroying the organisms in the nasopharynx of the carrier. If this could be achieved, a sudden diminution of the carrier rate would be expected and there would be an interval during which no further cases would occur. The carrier rate was determined on a sample population of the most severely affected battalion. Then all men of this battalion and carriers found in other units were given 2 tablets (1 Gm.) of sulfapyridine three times a day for two days. After one day the carrier rate among the sample population was again determined. As a control, the carrier rate in an untreated battalion was also determined. Although 70 treated and only 43 untreated men were examined, no carriers were discovered among the treated men, but 10 were found among the untreated. Of still greater significance is the fact that 17 of the treated men were previously proved to be carriers. Under normal conditions, according to Flack (1917), about 20 per cent of contact carriers are infective for less than two weeks and the remainder for periods up to and for longer than twelve weeks. The carriers whose swabs yield a profuse growth of meningococci tend to be more persistent in their spreading of infection. The majority of the carriers whom the authors treated were of this type, and the results of their bacteriologic investigation have to a certain extent been confirmed by subsequent clinical observations and experience. In the month that has elapsed only 1 case of meningitis has occurred in the battalion treated, an officer who did not receive the treatment contracting the disease. A workman in the lines of this battalion who did not receive the prophylactic treatment died of meningitis. During this period several cases of meningitis occurred among the men of the control battalion until they, too, received the prescribed course of sulfapyridine. On the basis of their investigations Gray and Gear conclude that mass dosage is an efficient means of controlling immediately an epidemic of cerebrospinal meningitis by reducing the carrier rate to negligible proportions and preventing the onset of the disease in those in whom incubation occurs. The effect of the drug alone cannot be permanent, but they believe that by taking ordinary hygienic precautions the chances of a recrudescence of the disease in epidemic proportions is remote. They recommend that in the event of a case of cerebrospinal meningitis occurring in a semipermanent military camp the carrier rate should be determined if possible, and if it exceeds 20 per cent, mass dosing with sulfapyridine is justified. The ordinary hygienic precautions must not be neglected.

J. A. M. A.

SARCOMA OF THE PAROTID GLAND WITH ASSOCIATED SYNDROME OF THE SPHENOID FISSURE AND FACIAL PARALYSIS. A. LOPEZ VILLORIA, Arch. venezol. Soc. de oto-rino-laring., oftal. neurol. **2**:68 (June) 1941.

Lopez Villoria described a case of sarcoma of the parotid gland with marked extension to the pharyngeal wall, the zygomatic and pterygomaxillary fossae, the sphenomaxillary fissure and, finally, the orbit. As a result of this extensive involvement, there was encroachment on the sphenomaxillary fissure, with the production of a group of symptoms which have been termed by the author the syndrome of the sphenoid fissure. This syndrome is evidenced by complete paralysis of all the motor nerves of the orbit, without any evidence of exophthalmos. The latter observation is one of the important purposes in the presentation of this case.

PERSKY, Philadelphia.

INFECTIOUS MONONUCLEOSIS IN A CHINESE SIMULATING LARYNGEAL DIPHTHERIA WITH LARYNGEAL OBSTRUCTION. K. H. CH'EN and C. T. TENG, Chinese M. J. **59**:116 (Feb.) 1941.

A Chinese boy 11 months old was taken into the hospital of the Peiping Union Medical College on the third day of his disease; he remained there until he was cured, about one month. The early symptoms were thought to be due to laryngeal diphtheria, since there were fever, hoarseness and respiratory difficulty. Such was not the case, however, and this case is of interest because it is the first instance of this disease in a Chinese known to have been thoroughly studied; also, the child had been given diphtheria antitoxin horse serum.

The sheep cell agglutination titer reached 1:320 on the fourteenth day of the illness (eleven days after the injection of the horse serum); it came down to 1:160 on the twenty-second day and had about disappeared by the sixty-fifth day. Twelve control patients were studied, and 8 of these were given horse serum in equal or larger amounts than that given to the patient with infectious mononucleosis. The highest titer among all 12 of the controls was only 1:40, occurring in 2 instances. The level of agglutinins, therefore, is apparently definitely higher in patients with infectious mononucleosis than in controlled patients given horse serum.

NEFF, Kansas City, Mo. [AM. J. DIS. CHILD.]

IMPAIRMENT OF HEARING IN A PATIENT WITH RECKLINGHAUSEN'S NEUROFIBROMATOSIS. HISAO AOYAGI, Oto-rhino-laryng. (Abstr. Sect.) **14**:9 (March) 1941.

The patient was a 24 year old woman. At the age of 2 years she already showed on her chest several soft pea-sized nodular growths, which became gradually larger as she grew older. At the age of 11 years she had drooping of the left upper eyelid and weakness of the muscles on the left side of the face. In two years there was slight exophthalmos with impairment of vision on the left. Up to this time her mental development was above the average, but on account of the disturbance of the eye she left high school. About this time she noticed the appearance on her body of an area of yellowish brown pigmentation about the size of a thumb. At the age of 17 years she suffered from intermittent left ulnar neuritis, and in three years the fingers on the left hand were drawn up like an eagle's claw. At the age of 22 she had bilateral tinnitus, which was soon followed by bilateral impairment of hearing, worse on the left. There was no nausea, vomiting or dizziness.

Examination revealed: five nodular masses on the trunk varying in size from that of a grain of rice to that of a bean; four pigmented areas; contracture of the fingers and thumb of the left hand; altered reaction of the left ulnar nerve to electric stimulation; slight motor disturbance of the left lower extremity; paralysis of the muscles of the left side of the face; left exophthalmos; impairment of vision and narrowing of the visual field on the left; absence of reaction to light and accommodation in the left pupil; slight papilledema on the left;

widening of the left internal auditory meatus and destruction of the petrous apex, revealed in a roentgen study by Stenver's technic; marked disturbance in vestibular function, with complete loss of hearing on the left and partial loss on the right. Biopsy of the tumor of the skin on the chest indicated neurofibroma.

In view of the aforementioned findings Aoyagi concluded that the case was one of Recklinghausen's disease involving the second to eighth cranial nerves and, in addition, the ulnar and great sciatic nerves. HARA, Los Angeles.

PNEUMOCOCCIC MENINGITIS TREATED WITH SPECIFIC SERUM AND CHEMOTHERAPEUTIC DRUGS OF THE SULFANILAMIDE GROUP. HENNING VOGELIUS, Acta med. Scandinav. **106**:449, 1941.

Since 1938, 21 cases of pneumococcic meningitis were observed in the Copenhagen Hospital. No recovery occurred until the use of sulfapyridine, in 1940. Since then 12 cases have been observed, in 4 of which there was recovery; in 3 of these specific serum was also given. The age in these cases was 12, 12, 13 and 22 years. In two thirds of these the origin of the infection was otorhinogenic; only in 1 case was the origin probably pneumonia.

KAJDI, Baltimore. [AM. J. DIS. CHILD.]

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Claude C. Cody, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Robert L. Moorhead, 125 Ramsen St., Brooklyn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James G. Dwyer, 375 Park Ave., New York.
President-Elect: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Edward J. Whalen, 750 Main St., Hartford, Conn.
Southern.—Chairman: Dr. Francis B. Blackmar, Swift Kyle Bldg., Columbus, Ga.
Middle.—Chairman: Dr. Voss Harrell, Hofman Bldg., Detroit.
Western.—Chairman: Dr. Irving M. Lupton, Medical Arts Bldg., Portland, Ore.

American Otological Society

President: Dr. Wesley C. Bowers, 17 E. 61st St., New York.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.